PULMONARY CONSUMPTION,

A NERVOUS DISEASE:

CONSIDERED AS SUCH FROM A PRACTICAL, A CLINICAL, AND A THERAPEUTIC STANDPOINT.

— BY —

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PREFACE.

This essay is an effort to give a rational account of the principal causes and of the nature of pulmonary consumption, and has been written in the belief that no theory of the origin of disease can ever earn the right of permanent existence if it falls short in pointing out the path through which the disease may be prevented or alleviated. It may be pertinently asked whether the bacillus theory of pulmonary consumption, which is so popular at present, has rendered any such service to medical science? Has it lessened the mortality rate of this disease in the past, or does it give any assurance of doing so in the future? Can any one claim that it has not been accorded a fair and generous hearing? Was ever a medical theory launched under more favorable auspices, or received with greater enthusiasm? Prompted by the hope, long deferred, that a knowledge of the tubercle bacilius would accomplish for phthisis what the germ idea had done for practical surgery, the medical profession, eagerly and frankly, accepted it, and thoroughly tested it; yet he who takes a calm and impartial retrospect of the whole situation must own that never was an *ignis fatuus* pursued which left more promises broken, and greater anticipations unfulfilled than the bacillus theory in so far as it stands related to the therapeutics of this disease.

With this conviction, the neurotic theory of pulmonary consumption, propounded in the following pages, is submitted to the critical consideration of the medical profession, in the belief that it explains most, if not all the varied and apparently opposing phenomena constantly observed in this disease, and also that it serves the practical purpose of pointing out the means and methods by which it is to be rationally and successfully treated.

1829 Spruce Street, Philadelphia, July, 1891.

CHAPTER I.

INTRODUCTION.

That pulmonary consumption is not due to one single cause, is but too apparent to anyone who takes a clear and an unbiased view of its origin. The general evidence already collected, shows pointedly enough that occupation, want of exercise, insufficient food, inheritance, excesses of all kinds, sex, order of birth, dampness, and change of climate are powerful factors in the production of this disease; and in carefully watching the progress of consumption one cannot help but be impressed with the belief that in many, and probably in all such patients, a disturbance of the nervous system plays either a causative or a concomitant rôle in its history.

The existence of such a relationship between consumption and the nervous system, had been faintly present in my mind for a number of years, but this did not assume a definite form until the year 1886, when the following striking case came under my personal supervision. In June of that year, Mrs. W., aged 26, became my patient with the following history: Confined the previous December, from which she made a good recovery with the exception of a violent attack of pain in the region of the right ovary, as she described it, about ten days after labor. She remained well, however, until the following January,

when she became chilly occasionally, and began to have night sweats. When I saw her she had lost some flesh, no hæmoptysis, poor appetite, irregular bowels, and a whitish expectoration, not at all profuse. Her menses reappeared one month before (May). She never had malaria, nor rheumatism, but she complained of an ill-defined and sometimes shooting pain throughout the body, which localized itself especially behind the right ear, right side of the neck, and right costal portion of the chest. She also perspired more profusely around the back part of the head and neck than anywhere else.

Physical examination showed body somewhat emaciated; heart normal. Right lung: Diminished respiratory motion throughout, and a slight impaired percussion resonance in apex, associated with a few mucous râles in same area. Left lung: Want of proper expansion, otherwise normal. There is a family history of the disease. She was also at the same time examined by an expert gynæcologist, who pronounced her generative organs sound. Under treatment her appetite, cough, and expectoration gradually improved, and the mucous râles had all disappeared by the following August; and, with the exception of several attacks of pain in the right ovarian region, which, during their greatest intensity, radiated upwards to the stomach and intestines, she apparently felt better. Her menses failed to appear after September, and from this time on, her case developed

the most uncommon nervous symptoms. Her temperature, which did not vary much from the normal hitherto, now began to rise. The ovarian pain became very much aggravated, and tenderness began to show itself along the whole spine. The pain in the right side of the head, neck, and chest, of which she complained at her first visit, also became worse. In November there was dulness in right apex, subcrepitation in same area, extending to third rib in front, and to angle of scapula behind. Evening temperature 101°. The ovarian pain appeared now at more frequent intervals, and nothing but large doses of morphine would relieve it. In December her arms and lower limbs became so painfully stiff, that she was only able to move them with the greatest difficulty. This attack lasted for four or five days, when it entirely disappeared. The pain in the ovarian region and that in the right chest often came on alternately. She frequently said that when she had pain in her right lung there was none in her abdomen, and *vice versa*. The application of a hot poultice to the chest would drive it to the parts below, and the reverse of this was also true. By applying one pole of a Galvanic battery between the shoulders and the other on the sternum, the pain would disappear at once from the chest, but would reappear in the pelvic region. Application of both poles along the spine—one in the cervical and the other in the sacral region—would remove the pain altogether. In

fact, the Galvanic current was the only means which afforded her any relief, and this was decided and instantaneous. After the manifestation of these nervous symptoms she became rapidly worse, and died in a short time. At no time was there any œdema or paralysis of the extremities in her case; and at no time, except when she was so stiff, did she suffer from any well-marked pain in these parts of her body.

The question which presents itself here for discussion is as to the precise nature of the relationship between the nervous and the lung affection: Was the one dependent on the other, or not? If so, which was primary, and which was secondary? Or were both morbid conditions manifestations of a still deeper-lying disorder? This is, of course, very difficult to determine; but it is very certain that, so far as time is concerned in this case, the nervous symptoms antedated those of the lung, and that the former aggravated the latter. The special interesting features of the case were, the confinement of the disease to one side of the body until up to within a short time of her death, and the close sympathy which existed between the pain in the right ovary and that in the right side of the chest. It seemed as if the pain in the right side of the head, neck, and chest, was reflex in character, and depended on the ovarian disturbance as its primary source of irritation, and that in time a reciprocal channel of communication was established between the two, through which they reacted the one on the other.

The literature on the subject of the association of pulmonary consumption and the nervous system, is, so far as my knowledge extends, very scant. Most writers on phthisis, and on diseases of the nervous system, ignore it entirely, while many of those who do recognize it strangely regard the two conditions as being antagonistic to each other. As early as the year 1850 Dr. J. C. Holland defined pulmonary consumption as "a disordered condition of the nervous system."* Dr. Copland looked upon tuberculosis and scrofula as depending on abnormal conditions of the nervous system; that the accompanying disturbances of digestion, of assimilation, of the circulation, and even the local determination of these diseases, he refers to the state of the nervous influence of these parts.† In the *Medical Times and Gazette*, 1871, vol. ii, p. 613, Dr. Clifford Allbutt,‡ under the heading of "Phthisis as a Neurosis," discusses a variety of phthisis which, he holds, does not agree with the general meaning of the term "phthisis." He says that patients belonging to this type of the disease come from the neurotic class, in illustration of which he cites the following cases which came under his observation:

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* "On the Nature and Cure of Consumption," cited from Ancell's "Treatise on Tuberculosis," p. 556.

† "Dict. Practice of Medicine," part xv, p. 750.

‡ See also by same author, "Neuroses of the Viscera," *Lancet*, 1884, vol. i, p. 604.

About a year ago I attended a refined lady, *belonging to a highly neurotic family*, and whose children presented like characters, she had been a nervous, irritable, neuralgic woman all her life, but never actually ill in any serious way. She was worn down by nursing one who was very dear to her, and whose death, which followed, shocked and prostrated her still more. She took to her bed with consolidation of the apex of the right lung, then of the left; in both, the mischief spread rapidly; hectic fever ran high; in three weeks she was dead. Her age was forty-three. On the autopsy, which was made in consequence of her death appearing to her family to be very sudden, we could not find a single tubercle in the body; but the apices of the lungs were almost destroyed, and less degrees of mischief were found below.

I had seen another case, a few years before, in which such a galloping consumption had occurred afrer nervous depression in a highly neurotic subject. The son of nervous parents on both sides, the father being odd and eccentric, and the mother actually insane, he was himself one of those heady, impassionable, gay fellows who make our charming prodigals—and a very pretty prodigal he was. Ruined in purse and character, and terribly depressed, though I do not think in any great degree worn out by actual vice, he arrived at home to meet with the reception such parents (or, at any rate, one of them) were likely to give him. A few days later he began to feel more and more exhausted; his pulse and temperature ran up; his right lung solidified and broke down; the left lung followed; and after five weeks of distressing illness he was dead. This case was the first which impressed me strongly with its probable neurotic origin. . . . . Having, then, cases of phthisis

presenting the exact features already described, and having also many cases of catarrhal broncho-pneumonic phthisis, and, more strictly, tubercular phthisis, presenting like character intercurrently with others of their own, I felt that neither the catarrhal nor the tubercular theory accounted for all instances . . . . . until I began to find, in my own practice and in the writings of alienists, how large a part phthisis plays in neurotic families. Even then, however, it did not occur to me to associate any particular form of phthisis with neurotic disorder, until a few striking cases of my unclassable variety occurred in neurotic families under circumstances which spoke too eloquently to be overlooked. . . . . If we try to go a step further, and ask for a pathological explanation of these facts, we approach a land of darkness. The more, however, I study the relations of the disease, the more I am satisfied that the lung mischief is also a neurosis—by which I mean, that the lesion is one not originating in the local tissues, but in the nervous system.

CHAPTER II.

RELATION BETWEEN INSANITY, HYSTERIA, HEADACHE, NEURALGIA AND CONSUMPTION.

It is not necessary to confine ourselves to generalities in considering the relationship between the nervous system and consumption, for a closer inspection will show us that special forms of nervous diseases, like insanity, hysteria, headache, and neuralgia are common accompaniments of this disease, as will be seen from the evidence contained in the present chapter.

In an extremely valuable contribution, entitled "The Pulmonary Pathology of General Paralysis,"* Dr. J. Crichton-Browne adduces testimony which proves that insanity and pulmonary disintegration, and especially pulmonary consumption, are closely enough interwoven to give grounds for believing that the former bears a causal relation to the latter. In this report he details the pulmonary changes which were present in the bodies of one hundred general paralytics after death, as follows:

Pulmonary congestion was most common, and present to a much greater degree in general paralysis than is found after slow death from exhausting bodily disease. In forty-nine out of one hundred cases in which it was present, it was so marked that it was diffi-

* See Brain, vol. vi., p. 317. 1883.

cult to say whether or not it was a pneumonic consolidation. In thirty-eight of the forty-nine cases it existed in both lungs, in eleven cases it was limited to the right lung, and in six to the left; and whether involving one or both *lungs*, it was invariably more extreme and extensively diffused in the male than in the female sex. The congestion of the lungs in general paralysis is of course hypostatic in character, and is almost invariably most marked posteriorly and in the lower lobes.

But in twenty-five out of the forty-nine cases it extended throughout the whole lung. The congestion was also associated with bronchial catarrh. In seventeen cases there was pus in the bronchii, in thirty cases frothy mucus, with increased vascularity of the bronchial mucous membrane.

Pneumonia was present in thirteen out of the one hundred cases. Two were acute and eleven were broncho- or hypostatic pneumonia. Both lungs were involved in eight cases, the right lung alone in four cases, and the left lung alone in one case. Twelve were males, and one a female. The fact that both lungs were inflamed in a large majority of the instances, shows that the pneumonia of general paralysis has a striking resemblance to the ordinary pneumonia found in practice.

Phthisis was found in twenty-five out of the one hundred cases.

In six of these only the remnants of past phthisical diseases were found in the lungs, which consisted of earthy or chalky matter, situated at the apex in

every case, occupying one apex in four and both in two cases.

These lesions undoubtedly occurred previous to the onset of the paresis. In the remaining nineteen cases the changes were recent. The lesions were confined to the right lung in three cases (males), to the left in seven cases (four males and three females), and to both lungs in nine cases (five males and four females). The disease consisted of gray miliary tubercles in nine cases; of caseous masses in five cases; and of mixed gray granulations and yellow cheesy masses in five cases. Cavities were associated with the miliary tubercles in three cases, and with the mixed tubercular and caseous degeneration in four cases. The vomicæ, which in five cases were confined to the upper and in one to the lower lobe, were always of small size, with ragged walls and without false membranes. There can be no question that in the nineteen cases the phthisis had arisen during the course of the general paralysis, and was cut short by the fatal termination of the latter.

Further evidence of the same kind is afforded in the statistical report of 1,240 *post-mortem* cases of insane subjects by Snell, jun. (*Ueber die Lungenschwindsucht bei Geisteskrankheiten.* Neurolgisches Centralblatt, 1887, s. 261), in which it was found that about 30 per cent. suffered from phthisis. On account of the fact that, as a rule, phthisis developed in the wake

of insanity, the author concludes that the latter acts as a predisposing cause to the former.

Dr. Walshe, in his "*Diseases of the Lungs*," 4th edition, p. 455, states that "hysteria is, on the whole, repulsive of phthisis as a coexistence—that is, if a woman with hysteria becomes genuinely phthisical, the nervous affection falls into abeyance," and again on p. 479, in qualification of the above, he says :

An hysterical woman may have frequent, shallow respiration, impaired percussion—tone, cough, expectoration, peculiar watery hæmoptysis, and night perspirations; she may lose flesh, and this even especially about the chest, and yet be non-phthisical. The existence of well-marked hysterical conditions of the spine, intercostal nerves, and skin, coupled with the peculiar watery hæmoptysis and the inordinate frequency of breathing, are valuable guides to the diagnosis. . . . . And, again, though hysteria and phthisis be, in the main antagonistic diseases, the antagonism is asuredly not absolute, and faith too confident in its reality has more than once led to serious error. It has every now and then happened that an hysterical woman, whose alleged chest-ailments were treated as pure figments, has died of genuine phthisis, while supposed to be simulating the disease.

Pidoux says that neuroses, hysteria, melancholia, the proteiform neuropathic state known as "neurosism," often act as moderating agents. Phthisis in neurotic subjects has a very slow development, and periods of long intermission. I have no doubt that

such subjects, among whom the disease is not rare, offer to it an extraordinary and almost indefinite resistance; and in treating them we must not pay too much attention to their abnormal nerves. . . . . If a pulmonary consumption is combined with a neurosis, the organic lesion will evolve very gradually; gastralgias and enteralgias have often this effect. I look upon this statement as an axiom ("*Études sur la Phthisie*," p. 151). And Largaud says:

We shall show how consumptives, even when most threatened, may live, thanks to hysteria; . . . . the neurosis appears to play the part of a protector, as if in its presence the pulmonary complaint had its symptoms mitigated and its progress arrested. . . . Hysteria in a consumptive is not a symptom of tuberculosis; it is not a complication; it is only a new morbid state without causal relation with it ("*Thesis*," Montpelier, 1882).

In a very interesting, and in some respects a remarkable series of papers on "*The Relation of Hysteria with the Scrofulous and the Tubercular Diathesis*," by Professor J Grasset, M. D., published in *Brain*, vol. vi, p. 433, and vol. vii, pp. 13 and 161, appear the following views in explanation of the relation between hysteria and phthisis:

Hysteria is the manifestation of the tubercular diathesis just as much as chorea is a manifestation of the rheumatic, and angina pectoris that of the gouty diathesis.

TABULAR VIEW OF DR. GRASSET'S CASES OF HYSTERIA.

No.	Sex	Age	Disease.	Family History.
1	F.	3	Hysteria	Father scrofulous, mother nervous and scrofulous, brother and sister died of cerebral disease.
2	F.	14	"	Mother died of consumption.
3	F.	27	"	Father foolish; one sister died of whooping-cough,
	F.	38		one of cerebro-spinal meningitis, and one of consumption.
4	F.	32	Hysteria and bronchitis.	Mother and brother died of consumption.
5	F.	10	Hystero-epilepsy.	Father died of pneumonia, mother of heart disease, two sisters of phthisis, and one is hysterical.
6	F.	9	Hysteria.	Mother died of phthisis, father well.
7	F.	29	"	Mother died of phthisis.
8	F.	7	"	One brother died of phthisis, two sisters always sick.
9	F.	24	"	Mother hysterical, father died of phthisis, one sister died of convulsions.
10	F.	22	"	Mother and sister died of phthisis.
11	F.	17	"	Mother and father died of phthisis.
12	F.	29	"	Mother and father died of phthisis.
13	F.	29	"	Mother, sister, and brother died of phthisis.
14	F.	22	"	Mother hysterical, father died of phthisis.

TABULAR VIEW OF DR. GRASSET'S CASES OF HYSTERIA (*Continued*).

No.	Sex	Age	Disease.	Family History.
15	F.	22	Hysteria.	Mother died of, and sister suffering from, phthisis.
16	M.	15	"	Father well, mother scrofulous, sister died of phthisis, and one brother of tubercular meningitis.
17	F.	20	Phthisis and hysteria.	Father and three sisters died of phthisis.
18	F.	22	Hysteria.	Father an inebriate and vomited blood, mother died of phthisis.
19	F.	25	"	Mother died of phthisis, father of insanity.
20	F.	42	"	Father died suddenly; mother of phthisis
21	F.		"	Mother died of phthisis, father insane, one brother had hæmoptysis.
22	F.	19	"	Mother scrofulous, father phthisical.
23	F.	11	"	Mother nervous.
24	F.	42	"	Mother died young of a protracted illness; uncle and aunt died of phthisis.
25	F.	35	"	Mother neurotic, father and brother died of phthisis.
26	F.	18	Hysteria and phthisis.	Father was a drunkard and died of pleurisy, mother bronchitic, maternal grandmother died of phthisis.
27	F.		Hysteria and phthisis.	Father was a drunkard and died of dropsy, mother had hæmoptysis and died of bronchitis, five sisters dead.

TABULAR VIEW OF DR. GRASSET'S CASES OF HYSTERIA (*Continued*).

No.	Sex	Age	Disease.	Family History.
28	F.		Hysteria and phthisis.	Mother nervous, father died suddenly, one sister died of phthisis.
29	M.		Hysteria followed by phthisis.	Mother died young, father died insane, sister is neurotic.
30	F.		Phthisis followed by hysteria.	Mother nervous, mother died of cancer, brother and sister died of chest disease.
31	F.	7	Phthisis succeeded by somnambulism and recovery	Mother and father died of phthisis.
32	F.	29	Hysteria followed by phthisis.	History unknown (foundling).
33	M.	25	Hysteria followed by phthisis.	Sister died of phthisis.
34	M.	21	Phthisis followed by hysteria.	Sister neurotic, brother died of convulsions.
35	F.	47	Hysteria and phthisis.	Mother neurotic.
36	F.	55	Hystero-epilepsy and phthisis.	Mother died of pneumonia.
37	F.	20	Hysteria succeeded by phthisis and recovery.	Mother well, father died of phthisis.

By this he does not mean that hysterical subjects suffer from tubercles, but that the scrofulous and tubercular diatheses are constitutional states found in a series of generations of the same family, and among

a certain number they are represented as neuroses. Thus, for instance, he says, in a phthisical family you will see, of the children, one dying from tubercular meningitis, another become an ordinary consumptive, and the third escape the diathesis; or the last may be neurotic, hysterical or a lunatic even. But, according to Professor Grasset, the last has escaped the hereditary diathesis in appearance only. He is tubercular, like the rest, though he has no tubercles anywhere. It is his neurosis which represents the diathesic affection. He details the records of thirty-seven cases of which a tubulated report is found below, in which hysteria was associated with pulmonary consumption, or with the tubercular diathesis, and on account of their intrinsic value, I beg to direct the reader's attention either to the original, or to their full quotation in my lecture on this subject, published in the *Therapeutic Gazette* for November, 1888.

The histories of these cases simply form an invaluable contribution to medicine, and so far as my knowledge goes it is the first systematic effort to show the intimate relation been hysteria and pulmonary phthisis. This much its facts establish beyond a doubt. From this it does not follow, however, that Dr. Grasset is correct in his primary assumptions when he states that hysteria and pulmonary consumption are but the manifestations of a still deeper-lying diathesis, which he calls the tubercular; nor does it follow that these two diseases frequently alternate in

intensity, or act antagonistically, because they are both believed to be rooted in this same diathesis. The erroneousness of these views, as well as that of the other, that hysteria is the only nervous state with which pulmonary consumption is associated, will, I think, become quite evident on close inspection of the factors involved in this problem, and which will be presented in the following pages.

The displacement of hysteria by consumption and *vice versa*, as is manifested in these cases is by no means evidence that the alternation is the consequence of an inherent antagonism between these two diseases. Such a substitution is no more remarkable than many similar phenomena which are constantly met in other departments of practical medicine. Do we not frequently see cases of pulmonary consumption in which the cough and expectoration improve after the onset of diarrhœa, or in which the two former symptoms become aggravated when the latter ceases? Is it not also true that the gravest symptom and physical signs of this disease will be moderated by the appearance of a delayed catamenial flow? or will be driven into entire temporary abeyance by the pregnant state? To account for these interchanges on the score of an underlying diathesis would be in no greater harmony with the state of affairs than it is to hold that hysteria and phthisis are the outcroppings of a still more fundamental disorder. The most consistent interpretation of these phenom-

ena, so far as I am able to see, is, that they occur in accordance with the well-known empirical, though well-established, law, which implies that no two pathological processes occur with equal intensity in the body at the same time; and that both hysteria and consumption are probably but a two-sided expression of a diseased state of the nervous system.

Consumption also exists largely among those who suffer from other forms of nervous diseases. Dr. Stevens, in his interesting work on *Functional Nervous Diseases*, p. 139, gives a tabulated view of the family histories of 100 patients who were affected with neuralgia, headache, neurasthenia, chorea, epilepsy, etc., and in whom marked errors of refraction were also found. These 100 patients represented a family membership of 510 persons, among whom prevailed the following diseases: Consumption, in 104, or 20.40 per cent.; cerebro-spinal meningitis, epilepsy, chorea, headache and other nervous diseases, in 114, or 22.35 per cent.; heart disease and rheumatism in 22, or 4.31 per cent.; and Bright's disease in 17, or 3.33 per cent.

In a paper on *Eye Strain as a Cause of Headache and Neuralgia**, by Dr. Ambrose L. Ranney, the author tabulates the histories of 46 patients who suffered from eye strain combined with headache and neuralgia, and who were in most instances relieved of their

* See the Medical Record, June 22d, 1889.

nervous diseases by correcting the errors of refraction. The same author, in another communication,† says:

Many diseases, which are to day commonly regarded as of bacterial origion, owe their development, in my opinion, to some *underlying cause that has impaired the nervous functions*, and thus rendered the patient peculiarly susceptible to deleterious atmospheric influences. This view is held by many others beside myself. It is gaining ground among the profession in England and France, and lately Dr. Thomas J. Mays, of Philadelphia, has had the temerity to discuss in two public lectures, whether phthists is not to be regarded as a pure neurosis.‡ Some points in his theory seem to me to be rather untenable; but many of his observations respecting the clinical association of the tuberculous predisposition with various marked neuroses are unquestionably accurate and in accord with well accepted facts.

I have personally given this subject considerable attention, because in my maternal ancestry phthisis has been extremely frequent, and the duration of life materially lessened thereby. I am also confident that a correction of a high degree of latent hypermetropia (whose existence was unsuspected until atropine was instilled into my eyes) marked a turning-point in my own physical state which has never ceased to be a cause for gratitude.

For many years I have carefully investigated the ocular condition of every patient who had cause to fear, as I had done in the past, the dreaded advent of pulmonary consolidation and softening. I have

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† The Medical Examiner (N. Y.), May, 1889.
‡ Therapeutic Gazette, November and December, 1888.

never yet encountered a case of typical phthisis in which 'eye strain' did not exist as a factor (more or less potent, in my opinion, in causing and hastening its development). I believe most firmly that had this factor been recognized early in life, before the eyes were employed in study and other occupations, and if all anomalies of refraction and of muscular equilibrium had been thoroughly rectified at that time, many of the hopeless sufferers from phthisis, that we all have met, would have escaped the disease, not because certain atmospheric germs would not still have constantly assailed them, but because the vigor of their constitutions would not have been so sadly impaired by a constant and useless expenditure of nerve-force as to unfit them to combat disease.

CHAPTER III.

TABULATED HISTORIES SHOWING THE RELATION BETWEEN DISEASE OF THE VAGI AND CONSUMPTION AND OTHER PULMONARY AFFECTIONS.

If it is a truism that no organ can preserve its integrity when its supplying nerve is disordered, then the principle is practically ignored in so far as the relation between the lungs and the vagi is concerned. Indeed, the bacillus maëlstrom has so completely swept everything before it that hardly a thought is given to this phase of the pulmonary problem at the present day. When we further reflect that during recent years it has been found that peripheral nerve disease may give rise to serious organic lesions; that this knowledge has thrown new light on many phenomena which were before believed to be inexplicable; and that section of the vagi in animals is followed by inflammatory changes in the lungs; we may well ask if it is not incumbent on us as scientific men to ascertain whether or not the pneumogastric nerves really share any responsibility in the evolution of pulmonary consumption.

The following tabulated histories of cases are interesting both from an experimental and a clinical standpoint, inasmuch as they show that all forms and phases of pulmonary disease are constantly called forth through the instrumentality of vagus disintegration.

NO.	AGE	SEX	DISEASE OR INJURY.	POST-MORTEM CONDITION OF LUNGS.	POST MORTEM CONDITION OF VAGI.	OBSERVER
1			Contusion of left side of neck by bullet.	Absence of respiration in left lung.	Left vagus degenerated.	Stromeyer. Maximen der Kriegsheilkunde, 1861, S. 116
2	8 mos		Convulsions.	Collapse of right lung.	Right vagus compressed by enlarged gland	Goodhart. Brit. Med. Jour., 1879, vol. I, p. 542.
3	49	F.	Carcinomatous tumor of neck. Extirpation.	Œdema.	Excision of vagus during operation.	Diebel. Clbt. f. Chirurgie, 1881, S. 748.
4	55	M.	Sarcoma of left side of neck Removal.	Œdema.	Exsection of left vagus during operation.	S. W. Gross, private communication.
5	65	M.	Carcinoma of left neck Removal.	Œdema.	Left vagus flattened by tumor.	v. Langenbeck. Arch. f. K Chururg. Bd. I, S. 77.
6	Adult	M.	Sarcoma of neck. Removal.	Œdema.	Division of right vagus.	Busch Med. Times and Gaz., 1851, vol. 2, p. 176
7	Adult	M	Myxo-fibroma of left parotid. Operation.	Œdema and congestion.	Left vagus stretched.	Heath. Trans. Lond. Path. Soc., vol. 35, p. 342.
8	54	M.	Aortic aneurism.	Pleurisy and hæmorrhagic infarcts.	Left vagus flattened by pressure.	Smith. Lancet, 1888, p. 1022.
9	53	M.	Epilepsy.	Hæmorrhagic infarcts.	Disease of right vagus.	Stackler. Clbt. f. K. Med., 1883, p. 316.
10	10	M.	Swelling in left neck.	Hæmorrhagic infarcts in lungs.	Peri-neuritis of left vagus	Sommer. Char. Annalen., 1888, S. 647.
11			Scarlatina and glandular enlargement of neck. Operation.	Purulent bronchitis.	Exsection of left vagus.	Riedel. Fortschritte der Med., 1883, S. 499.
12	53	M.	Bronchitis.		Left vagus imbedded in lymph-gland, and was found to be atrophied.	Riegel. Berliner K. Wochenschrift, 1875, No. 31.
13	68	F.	Stabbed in the neck.	Pulmonary congestion and bronchitis.	Left vagus divided	Fearn. Amer. Jour. of Med. Sciences, 1848, p. 266.
14	46	M.	Glandular tumor of neck. Operation.	Bronchitis.	Vagus compressed by tumor.	Eccles. Lancet, vol. 1, 1844, p. 724.

NO.	AGE.	SEX.	DISEASE OR INJURY.	POST-MORTEM CONDITION OF LUNGS.	POST-MORTEM CONDITION OF VAGI.	OBSERVER.
15		M.	Injury to right vagus.	Latent pneumonia.	Right vagus wounded.	Verga. Allgemeine Chururgie der Kriegswunden nach Erfahrungen in den Nord-Italianschen Hospitalern, Bd. 2, S. 83.
16	45	M.	Cancer of œsophagus.	Pneumonia.	Right vagus implicated.	Gull. Guy's Hospital Rep. 3d series, vol. v, 1859 p. 307.
17	55	F.	Encephaloid tumor of post-mediastium.	Pneumonia.	Right vagus imbedded.	Quain. Trans. Lond. Path. Soc, 1857, p. 45.
18	27	M.	Enlarged bronchial gland.	Pneumonia and pleurisy.	Left vagus compressed.	Maixner Prager Vierteljahrschrift, 1879, 1. S. 87.
19	33	M.	Pneumonia.		Vagus compressed by small tumor.	Weil. Archiv. f. K. Med., Bd. xiv, 1876.
20	8	M.	Enlarged bronchial gland.	Broncho-pneumonia.	Right vagus compressed by gland.	Goodhart. Brit. Med. Jour., 1879, vol. i, p 542.
21	38	M.	Syphilis.	Pneumonia.	Vagus imbedded in tumor.	Unverricht. Neurologisches Clbt., 1888, p. 164.
22	47	M.	Pneumonia.		Right vagus atrophied and compressed by enlarged gland.	Pelizaeus. Inaugural Diss. Wurzburg, 1880.
23	31	M.	Tumor in cerebellum.	Gangrene and pneumonia.	Vagus compressed by tumor.	Habrich. Arch. f. Psych. u. Nervenkeilkunde, Bd. v, S. 500.
24	Adult	M.	Aneurism of innominate artery.	Broncho-pneumonia.	Right vagus flattened and degenerated.	Lécorche. Clbt. f. K. Med., vol. i, S. 282.
25	55	M.	Aneurism of innominate artery.	Pneumonia.	Vagus compressed by aneurism.	Hewson. Penn. Hospital Reports, 1868, p. 219.
26	56	M.	Aneurism of innominate artery.	Bronchitis.	Right vagus flattened and compressed.	Fergusson. Amer. Jour. of Med. Sci., 1842, vol. 3, p. 221.
27	72	M.	Cancer of the œsophagus.	Pneumonia.	Left vagus implicated in cancer.	Wilks. Trans. Lond. Path. Soc., vol. x, p. 159.
28	45	M.	Aneurism of aortic arch.	Pneumonia.	Left vagus compressed and atrophied.	Johnson. Trans. Lond. Path. Soc., p. 42, 1873.
29	Adult	M.	Mediastinal tumor.	Pneumonia.	Compression and degeneration of vagus.	Eger. Arch. f. Klin. Chururg., Bd. 18, S. 502.

NO.	AGE.	SEX.	DISEASE OR INJURY.	POST-MORTEM CONDITION OF LUNGS.	POST-MORTEM CONDITION OF VAGI.	OBSERVER.
30	69	M.	Aneurism of arch of aorta.	Phthisis.	Left vagus compressed by aneurism.	v. Ziemssen. v. Ziemssen's Handbuch, Bd. xi, S. 2.
31	Adult	M.	Aneurism of arch of aorta.	Phthisis.	Right vagus compressed by aneurism.	Bernheim and Simon. Internationales Clht. f. Laryng., 1887, 1888, S. 68.
32	35	M.	Aneurism of arch of aorta.	Phthisis.	Left vagus compressed by aneurism.	Gull. Guy's Hosp. Rep., 3d series, vol. v, 6859, p. 307.
33	61	M.	Mediastinal tumor.	Phthisis.	Right vagus compressed by aneurism.	Gull Ibid., p. 312.
34	19	F.	Tachycardia.	Phthisis.	Vagus compressed by enlarged bronchial glands.	Guttmann. Virchow's Arch., Bd. 59, S. 51.
35	18	M.	Tachycardia.	Phthisis.	Left vagus compressed by enlarged glands.	Mercklen. Deutsche Medizinal Zeitung, 1887. S 1108.
36	33	F.	Tachycardia.	Phthisis.	Vagus compressed by enlarged cervical glands.	Angyan. Wien. Med. Wochenschrift, 1884, S. 515.
37	Adult	F.	Tachycardia.	Phthisis.	Carcinomatous degeneration of right vagus.	Arnold. Deutsches Archiv f. K. Med., Bd. vi, S. 277.
38	3	F.	Dyspnœa.	Not stated.	Compression of right vagus by gland.	Chapin. N. Y. Med. Jour., 1884, p. 294.
39	43		Tumor of parotid gland.	Phthisis.	Vagus imbedded, swollen and red.	Pilz Arch. f. Klin. Chururg., Bd. 9, S. 336.
40	46	M	Dilatation of aortic arch.	Phthisis.	Right vagus degenerated and compressed.	Stimson. Amer Jour. Med. Sci., 1881, p. 192.
41	58	M.	Epithelial carcinoma on right side of neck. Extirpation.	Phthisis.	Right vagus compressed.	Langeubeck. Arch. f. Klin. Chururg., Bd. i, S. 73.
42	43	M.	Malignant cyst of the neck.	Phthisis.	Left vagus imbedded in tumor.	Treves. Trans. Lond. Path. Soc., 1887, p. 367.
43	55	M.	Aneurism of the aorta.	Phthisis.	Left vagus compressed.	Whipham. Ibid., 1882, p. 82.
44	20	M.	Cancerous tumor of mediastinum.	Phthisis.	Right vagus implicated.	Murchison. Ibid., vol. x, p 240.
45	53	M.	Aneurism of the inuominate.	Phthisis.	Right vagus compressed.	Baümler. Ibid., vol. 23, p 66.
46	59	M.	Cancer of œsophagus.	Phthisis.	Both vagi implicated.	Hanot. Archiv Geuerale de Med. Tom, 28, 1876.

NO.	AGE.	SEX.	DISEASE OR INJURY.	POST-MORTEM CONDITION OF LUNGS.	POST-MORTEM CONDITION OF VAGI.	OBSERVER.
47	38	M.	Aneurism of aortic arch.	Phthisis.	Compression of right vagus.	Hauot. Ibid., p 297.
48	7 mos	M.	Measles.	Phthisis.	Right vagus compressed by bron. gland	Barlow. Trans. Lond. Path., Soc., vol. 30, p. 254.
49			Phthisis.	Phthisis.	Vagus inflamed and compressed by bronchial glands.	Grocco. Schmidt. Jahrbücher, Bd. 214, S 29.
50	38	M	Carcinoma of thyroid cartilage.	Phthisis.	Author believed that right vagus was implicated	Sbech. Arch. f. K. Med , vol. xxiii, p. 2
51	28	M.	Tachycardia.	Phthisis.	Author believes vagus was compressed by enlarged glands.	Guiter. Virchow und Hirsch, 22, 1887, Th. i, S. 203.
52	53	M.	Angina pectoris.	Not stated.	Right vagus compressed by enlarged glands	Leroux. Clbt. f. Nervenheilkunde, vol i, p. 251.
53	28	M.	Multiple neuritis.	Hæmorrhagic infarcts in lungs.	Vagi diseased.	Putnam. Bost. Med. and Surg. Jour., Feb. 14th, 1889, p. 159.
54	31	M.	Tumor of medulla oblongata.	General bronchitis.	Nucleus of vagus implicated.	Glynn. Liverpool Medico-Chirurg. Jour., vol 7, p. 428.
55	8	M.	Diphtheria.	Bronchitis.	Vagus degenerated	Mendel. Clbt. f. Nervenheilkde, Bd. 8, S 102.
56	30	F.	Bulbar paralysis.	Brouchitis. (Recovery.)	Vagus degenerated	Adamkiewicz Ibid., Bd. 3, p 168.
57	15	F.	Tachycardia.	Pneumonia.	Author believed the vagi diseased	Löwitt. Clbt. f. d. Med. Wis., 1880, p. 61.
58	Adult	F.	Syphilis.	Pneumonia	Both vagi atrophied	Penzoldt Ibid., 1874. S. 474.
59	7		Diphtheria.	Pneumonia.	Fatty degeneration of vagi.	Shech Deutsch. Arch. f K. Med., Bd. 23, S. 2.
60	18	M.	Multiple neuritis.	Pneumonia.	Left vagus grayish red and inflamed	Freud. Wiener Med. Wochenschrift, 1886, S 168.
61	31	M.	Multiple neuritis.	Pneumonia.	Vagi not examined	Stewart. Ed. Med. Jour., April, 1881, p 86[illegible].
62	30	M.	Gliomatous tumor in 4th ventricle.	Pneumonia.	Vagus centre compressed.	Schmidt Jour. of Nervous and Mental Diseases, 1887, p 82.

NO.	AGE.	SEX.	DISEASE OR INJURY.	POST-MORTEM CONNITION OF LUNGS.	POST-MORTEM CONNITION OF VAGI.	OBSERVER.
63	58	F.	Spinal Sclerosis.	Broncho-pneumonia.	Left vagus atrophied.	Stadelmann. Deutsches Arch. f. K. Med., Bd. 33, S. 125.
64	47	M.	Bulbar paralysis.	Broncho-pneumonia.	Vagus roots degenerated.	Freud. Deutsches Arch. f. K Med., Bd. 37, p. 405.
65	13	F.	Degenerative neuritis.	Pneumoula.	Vagi degenerated.	Kast. Ibid., Bd. 40, S. 41.
66	48	F.	Bulbar paralysis.	Broncho-pneumonia.	Vagus degenerated	Virchow's Archiv., Bd. 61, p. 1.
67	34	M.	Tumor of pons.	Phthisis.	Nothing stated about vagi.	Neurologisches Clbt., Bd iv, 1884, S. 361.
68	Adult	F.	Phthisis.	Phthisis.	Both vagi diseased	Bignardi. Longet. Anatomie u. Phy. des Nervensystems, Bd. 2, S. 313.
69	35	M.	Acute multiple neuritis.	Phthisis.	Marked degeneration of vagi.	Rosenheim. Archiv f. Psych. u. Nervenkrank., Bd. 18, S. 782.
70	42	M.	Multiple neuritis.	Phthisis.	Vagi degenerated.	Vierordt. Ibid., Bd. 14, p. 678.
71	48	M.	Multiple sclerosis	Phthisis.	Vagi not examined	Koppen Ibid., Bd. 17, S. 63.
72	40	M.	Cerebro-spinal sclerosis.	Phthisis.	Medulla degeuerated.	Ibid.
73	42	F.	Cerebro-spinal sclerosis.	Phthisis.	Vagus roots diseased.	Ibid.
74	25	F.	Neuromata.	Phthisis.	Both vagi diseased.	Wilks. Trans. Lond. Path. Soc., vol. x, p. 1.
75	23	M.	Neuromata.	Phthisis.	Both vagi diseased.	Hensinger. Virch. Archiv., vol. 27, S. 206.
76	11	M.	Tumor of pons.	Phthisis.	Nucleus of right vagus diseased.	Bleuler. Deutsch. Archiv f. Klin. Med., Bd. 37, S. 527
77	35	F.	(Syphilis). Multiple neuritis.	Phthisis.	Vagi degenerated.	Vierordt. Arch. f. Psych. u Nervenk., Bd. 14, S. 678.
78	Adult	F	Multiple sclerosis	Phthisis.	Nuclei of vagi degenerated.	Guttmann. Zeitschrift f. K. Med., vol. 2, S. 46.
79	Child	F.	Measles and swelling of mediastinal glands.	Phthisis.	Right vagus compressed.	Basevi. Jahrbuch. f. Kinderheilkunde, 1878, S. 414.
80	41	M.	Tabes dorsalis.	Phthisis.	Right vagus degenerated.	Oppenheim. Archiv f. Psych. u. Nervenk., Bd. 18, S. 125.

NO.	AGE.	SEX.	D SEASE OR INJURY.	POST-MORTEM CONDITION OF LUNGS.	POST-MORTEM CONDITION OF VAGI.	OBSERVER.
81	39	M.	Tabes dorsalis.	Phthis's.	Atrophy of right vagus.	Ibid., S. 145.
82	33	M.	Bulbar paralysis.	Phthisis.	Vagus root degenerated	Eiseulohr. Ibid , Bd. 19, S. 34.
83	22	M.	Multiple neuroma.	Phthisis.	Vagi diseased.	Generisch. Virch. Arch., Bd. 49, S. 15.
84	28	F.	Peripheral neuritis.	Phtbisis	Vagi diseased	Pitres et Vaillard. Revue de Med., 1886, p. 193.
85	Adult	F.	Tabes dorsalis.	Phthisis.	Vagus diseased.	Oppenheim. Arch. f. Psych. u. Nervenkrank, Bd. xx, S. 1
86	27	M.	Multiple neuritis.	Phthisis (Recovered.)	Vagi not examined	Senator. Zeitschrift f. K. Med., Bd. xv, S. 61.
87	70	F.	Tachycardia.	Infiltration of apices	Author believes vagi to be diseased.	Roth. Clbt. f. K. Med., 1885, S. 294.
88	38	F.	Tabes dorsalis.	Phthisis. (Recovered)	Peripheral nerves degenerated. Vagi not examined.	Sakaky. Arch. f. Psych. u. Nervenkrank , Bd. 15, S. 584.
89	31	M.	Tachycardia.	Phtbisical.	Author believes vagi to be at fault.	Roseubach. Clbt. f. K. Med , Bd. 3. S. 40
90	45	M.	Cancer of œsophagus.	Sloughing pneumonia.	Vagi involved.	Habershon. Diseases of Abdomen, 3d Ed., p 90.
91	63	F.	Cancer of œsophagus.	Pleuro-pneumonia.	Vagi diseased.	Ibid , p. 92.
92	71	M.	Cancer of œsophagus.	Pneumonia	Vagi diseased.	Ibid., p. 96
93	51	M.	Cancer of œsophagus.	Pneumonia.	Vagus diseased.	Ibid., p. 97.
94	53	M.	Cancer of œsophagus.	Phthisis.	Vagi involved	Ibid., p 98.
95	45	M.	Cancer of œsophagus.	Sloughing pneumonia.	Vagi involved.	Ibid., p. 100.
96	63	M	Cancer of œsophagus	Pneumonia.	Vagi involved.	Ibid., p. 82.
97	50	M.	Cancer of œsophagus.	Pneumonia	Vagi involved.	Ibid., p. 82.
98	48	F.	Bulbar paralysis	Pneumonia.	Nuclei of vagi diseased.	Remak. Neurologisches Centralbtt., 1888, p. 62

CHAPTER IV.

RELATION BETWEEN ALCOHOL AND CONSUMPTION.

Besides the evidence which has been gathered in the last chapter in regard to the intimate association existing between vagus disintegration and pulmonary disease, further testimony will appear in the following pages to show that there are certain other specific agencies, which, by destroying the potency of the pneumogastric nerves, also have the power of producing pulmonary consumption. First among these I would place alcohol, not because it is more potent than any other causes, but because its action in this particular has been more thoroughly investigated. It goes without saying that the existence of an intimate relation between alcoholism and phthisis has long been suspected by those who are most familiar with these diseases. Let those who have any doubts on this point cast a retrospect over the family histories of the cases of consumption within their experience, and ascertain how often alcoholism prevailed either in the immediate or remote ancestry of their patients. It is evident that this condition obtains quite largely among those who frequent our hospitals and dispensaries, and there is reason for believing that the evil is not confined to this class alone. In some of the districts of Paris, Dr. Alison found that

about one-third of those who suffered from consumption were also inebriates.

The following eleven* instances, which have been culled from the extensive experience of my friend, Dr. T. D. Crothers, of Hartford, Conn., illustrate the truth of this connection:

J. B., aged 42 years, began the excessive use of spirits after the death of his wife. He was a merchant, temperate, prosperous, and a man of character. He became a steady drinker, and was practically intoxicated all the time. After an attack of delirium tremens, he was placed under my care. During the four months while under treatment, he was alternately depressed and elated. He complained of wandering pains, and changeable appetite, as well as of spasmodic periods of coughing. A few months after he left me, he relapsed and continued to drink until he died a year later.

His mother and two sisters died of consumption. His father died from injury, but his grandfather was asthmatic, and used spirits to excess for years. One uncle on his father's side died from excess of drink, and another one died of consumption. One uncle died from phthisis after many years of drink excess.

His grandfather on his mother's side drank more or less all his life, and died from some rheumatic trouble.

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*Journal of Inebriety, April, 1890

B. A., aged 35, a mechanic, began to use spirits for insomnia and general debility, and finally became a periodical inebriate. He was under treatment for six months, and recovered. His father, grandfather, and two uncles died of consumption. His mother was hysterical, and his grandmother on his mother's side died of some lung trouble. One brother died from chronic alcoholism, and a sister is a drug-taker.

C. H., age 48, an army officer, began to drink during the late war. He is now a dipsomaniac, with distinct free intervals of three months. His mother died of consumption two months after his birth, and his two sisters died of the same disease. His father's family is temperate, but several members have died of consumption. His grandfather on his mother's side was a sailor, and drank to excess at times.

E. P., age 38, a farmer. His drinking seems to date from a nervous shock following the burning of a barn by lightning. His two brothers are chronic inebriates, one sister is a morphine taker, and the other uses both spirits and drugs to excess for all kinds of imaginary evils. On his mother's side, a grandfather and three aunts and one uncle died of consumption. His mother is still living. His father died of pneumonia, and his grandmother on his father's side died of consumption.

E. J., age 31, a clerk of inferior mental and physical development, began to drink at puberty. Consumption has been the common family disease of both

parents. On his mother's side both consumption and inebriety have been common. On his father's side consumption alone has prevailed.

P. O., age 28, is without business, and drank from infancy. He is now a chronic inebriate and has had delirium tremens. His father and two uncles died of consumption. His mother is a woman of wealth and fashion, and she lost her mother and one sister from consumption.

M. B., a lawyer, 54 years old, who began to drink at fifty from no apparent cause. His father and grand father died of consumption, at fifty years of age.

D. T., age 38, a conductor, began to drink after an injury to the spine. A brother, who was injured at the same time, died of consumption. The mother and a sister, the grandfather, and grandmother, on his father's side, all died of consumption.

D. B., 24 years old, and without business, began to drink at puberty, and is now a chronic inebriate. Both parents died of consumption. His grandfather on his father's side, and two uncles on his mother's side, died of the same disease.

A. H., 34 years old, a physician, took morphine for malarial poisoning, and then used alcohol to great excess. His mother, and three uncles on his father's side, died of consumption. His older brother became an inebriate at about 30 years of age, and one sister is in Colorado to prevent consumption.

(*Quarterly Journal of Inebriety*, Oct., 1888, p. 390): G. U., came from England in 1798, and settled at New Haven, Conn. He was a harness-maker, a beer-drinker, and after middle life drank rum to excess, until death at 61 years of age. His wife was a healthy woman, and lived to 80 years of age. Eight sons grew to manhood and married. Six of them died of consumption under 45 years of age; one was killed by an accident; and one died from excessive use of spirits. Two daughters grew up and married; one died of consumption, the other in child-birth. They left four children; two were inebriates, and the others were eccentric and died of consumption. Of the children of the eight sons, only ten grew up to manhood; four of these drank to excess and died; three of the six remaining died of consumption, and two others were nervous invalids until death in middle life; the last one, a physician of eminence, has become an inebriate, and is under care at present—he is the only surviving member of all this family. The male members of this family were farmers, tradesmen, and men of more than average vigor in appearance. They married women (so far as can be ascertained) without any special hereditary history of consumption or inebriety.

(*Ueber die Trunksucht und ihre Erblichkeit, von* Dr. J. Thomsen, Archiv. f. Psychiatrie u. Nervenkrankheiten. Band 17, 1886. Seite 536) abstract: Father was an inebriate until after he was 40 years old, at which time a cardiac affection developed itself from which he ultimately died, but which had the power of restraining him from exercising his morbid appetite. His brother was a drunkard too. Three of

his sons became confirmed alcoholics; one daughter died of phthisis; and another son died of general paralysis.

Dr. Grasset. (*Scrofulous and the Tubercular Diathesis*, Brain, vol. 7, p. 19) condensed: Father violent, an alcoholic and a libertine. Mother very nervous, and died of cancer of the uterus. Many of patient's relations are drunkards. Her brother and sister died of chest disease, and another brother is always ill and coughs a great deal. She was admitted May 5, 1879. One month previously she had a chill, rigors, and feverishness, which confined her to bed for four days; then she began to cough, and had two copious hæmoptyses. She sweats profusely at night, is losing flesh, and, in a word, has all the symptoms of pulmonary phthisis. Physical examination shows evidence of tuberculosis of both apices.

The histories of these cases give the most unmistakable proof that alcoholism and phthisis are not mere coincidences, but that they have a relationship so intimate that one may be converted into the other. The problem arises now as to the channel through which alcohol produces this disease, and this I believe resides in the pneumogastric nerve. For whatever else may be said of the action of alcohol, it is pretty generally understood that it possesses a special affinity for the nervous system, and that it produces its principle ravages in the body by operating on this, and by preference on the peripheral nervous tissue. Dr.

James Jackson, in this country, and Dr. Wilks, in England, were, I believe, the first to point out this form of disease, and they called it alcoholic paralysis. It has since then received the more appropriate name of alcoholic neuritis, and it is characterized in its early stages by numbness, tingling, hyperæsthesia in the extremities, and later on by anæsthesia, paralysis of motion, loss of knee jerk, quickened pulse, shortness of breath, and frequently by pulmonary embarrassment. The brain and spinal cord remain comparatively normal. The morbid changes occurring in the peripheral nerves under the influence of alcohol are parenchymatous and interstitial, or in other words they are confined to the nerve substance itself, or to its investing membrane. As a rule these changes occur together, the latter in many instances depending on the former, but frequently one exists exclusively of the other; especially is this true of the degeneration of the nerve fibre itself.

It being established, then, that alcohol has the power of degenerating the nerve fibres, it does not require a reckless flight of fancy to see how, by operating on the same tissue, it may bring about that peculiar destruction of lung substance known as pulmonary consumption. Degeneration of a nerve implies degeneration of the organ which it supplies with sensation and motion. Thus, degeneration of the sciatic nerve is followed by impairment of sensation and motion in the muscles and other textures of

the leg—a condition which is almost constantly present in chronic alcoholism, and degeneration of the pneumogastric nerves is just as naturally followed by disease of the lungs, heart, stomach, and all other organs supplied by them. This is no more than we may legitimately anticipate; for it has been proved already that division of, and protracted pressure of tumors, aneurisms, etc., on the pneumogastric nerves are capable of calling forth all the destructive lesions of pulmonary phthisis.

NO.	AGE.	SEX.	DISEASE OR INJURY.	POST-MORTEM CONDITION OF LUNGS.	POST-MORTEM CONDITION OF VAGI.	OBSERVERS.
99	Adult	M.	Delirium tremens.	Pneumouia.	Vagi not examined	Oppenheim and Siemerling. Arch. f. Pysch. u. Nervenkrank, Bd 18, S. 507.
100	45	F.	Chronic alcoholism	Phthisis.	Vagi not examined	Ibid., S. 506.
101	46	F.	Chronic alcoholism.	Pneumonia.	Vagi degenerated.	Déjerine Deutsche Med. Zeitung, 1887, S. 711.
102	39	M.	Diabetes and chronic alcoholism.	Not stated. (Death caused by paralysis of respiration.)	Vagus degenerated	Schultze. Neurologisch. Clbt., 1887, S. 271.
103	47	M.	Chronic alcoholism.	Phthisis.	Vagi and phrenici degenerated.	Strümphell. Arch. f. Psych u. Nervenkrank., Bd. 14, S. 339
104	26	M.	Chronic alcoholism.	Phthisis.	Medulla degenerated.	Oppenheim and Siemerling. Ibid., Bd. 18, S 114.
105	30	M	Chronic alcoholism.	Phthisis.	Medulla degenerated.	Vierordt. Neurologisches Clbt , 1886, S. 421.
106	Adult	F	Chronic alcoholism.	Phthisis.	Vagi and phrenici degenerated.	Sharkey. Trans. Lond. Path. Soc , 1888, p. 27.

The following tabulated cases will serve to illustrate the close anatomical and physiological association of chronic alcoholism and phthisis, as well as other destructive pulmonary changes with degeneration of the vagi and of the respiratory center (the latter of which is practically followed by the same effect), and with that of the peripheral nerves. The difficulty encountered in this research has not been so much in obtaining an abundance of material in which phthisis was evidently the direct result of alcoholic abuse, as it has been in finding the records of cases possessing all the points which I desire to emphasize in this essay, viz.: the coëxistence of pulmonary disintegration, alcoholism, and nerve degeneration, well brought out by a thorough *post mortem* demonstration.

CHAPTER V.

RELATION BETWEEN SYPHILIS AND CONSUMPTION.

A form of phthisis commonly called syphilitic has been recognized by most writers on pulmonary diseases. Indeed, it is a question with some whether or not all that which goes under the name of pulmonary cousumption and scrofula is really and originally due to syphilis. While this assumption is far too sweeping to be true, there can be no question that in the majority of instances in which phthisis has been produced through acquired syphilis, the symptoms, physical signs, and cause of the disease are peculiar, and distinguish it somewhat from other forms of phthisis.

Dr. A. Sokolowski (Ueber die Luetische Phthisie, Cent. f. d. Med. Wis., 1884, p. 122) says that, clinically, there are two distinct forms of syphilitic phthisis: (1) There appears, without any apparent signs of syphilis in other organs, well defined syphilitic hyperplasia in the lungs, with the signs of consolidation, etc., but with complete absence of the ordinary symptoms of phthisis, such as fever sweats, diarrhœa, etc.: (2) Materially more frequent is that form which manifests itself as syphilitic pneumonia, and in connection with syphilitic affections in other organs. In this form are present many of the characteristic signs of ordinary phthisis, such as wasting, cough, diarrhœa,

albuminuria, etc., but fever and sweats are likewise absent. Dyspnœa is frequently a prominent symptom, which may become asthmatic in character. At the autopsy he found in one case hard, cicatricial swellings, together with cavities in both apices; while in two other cases there was present, besides cavities, in the upper lobes, a well marked hyperplasia of connective tissue in other parts of the lungs. Dr. A. Hiller (Ueber Lungensyphilis und syphilitische Phthisie, *Charité Annalen* ix., 1884, s. 184) tabulates the post-mortem results of 58 cases of pulmonary syphilis. The typical changes which were found to be most abundant were interstitial connective tissue proliferation, peri-bronchial fibrous induration, diffuse thickening of the lobular parenchyma, and bronchopneumonia.

From these data it may be observed that syphilitic phthisis does not differ materially in its anatomical aspects from the non-specific, or ordinary variety. On this point Walshe ("Diseases of the Lungs," 4th ed., p. 512) also says:

There seems to be nothing distinctive in the anatomical character of the syphilitic variety of the product, and the connection between the low, quasi-inflammatory process producing it, and syphilis is only to be established by the antecedents of the individual and the coexistence of various positive results of tertiary syphilis.

Now, how does syphilis produce pulmonary consumption? Syphilis, like alcohol, unquestionably has

a specific affinity for the nervous system, and I believe that sufficient evidence will be found in the following tabulated cases to show that it, like alcohol, produces the lung-lesions through the pneumo-gastric nerves:

NO.	AGE.	SEX.	DISEASE OR INJURY.	POST-MORTEM CONDITION OF LUNGS.	POST-MORTEM CONDITION OF VAGI.	OBSERVERS.
107	35	F.	Multiple neuritis.	Phthisis.	Vagi diseased.	Vierordt. Arch. f. Psych. u. Nervenkrank., Bd. 14, S. 678.
108	22	M.	Paralysis of cranial nerves.	Pneumonia. (Recovered)	Author believes the vagi were diseased.	Lewtas. Med. Times and Gaz , 1865, vol. 2, p. 17.
109	19	F.	Syphilis.	Phthisis.	Medulla diseased.	Naunyn. Arch. f. K. Med., Bd. 34, S. 433.
110	29	F.	Syphilis.	Phthisis.	Medulla diseased.	Buss. Ibid., Bd. 41. S. 241.
111	30	M.	Paralysis.	Phthisis.	Medulla diseased.	Eisenlohr. Clbt. f. Nvukeilkunde, Bd. 10, S. 12
112	29	M.	Paralysis.	Phthisis.	Medulla aud cranial nerves diseased	Kahler Prager Zeitschrift f. Heilkunde, Bd. 8, S. 1.
113	35	M.	Syphilis.	Phthisis.	Left vagus diseased.	Berger. Deutsch. Arch f. K. Med.
114	23	F.	Paralysis.	Phthisis.	Right vagus diseased.	Vieroidt. Arch f. Psych. u. Nvnk., Bd. 14

These data show, then, very clearly that both syphilis and alcohol have the power of producing phthisis by impairing the pneumogastric nerves; and if one had the inclination to moralize on this subject, it would be very interesting to inquire why the North American Indian and other savages were practically free from pulmonary consumption until they came in contact with the white race? When we consider that

alcohol and syphilis are the greatest curses which the Indian has acquired from his white civilizer, I think it must be obvious that these two causes are largely responsible for sowing the seeds of pulmonary phthisis among these people.

CHAPTER VI.

RELATION BETWEEN EPILEPSY AND CONSUMPTION.

Epilepsy has long been recognized as an intimate associate of pulmonary consumption. The experiments of Van der Kolk, Marshall Hall, Brown-Séquard, and Kussmaul and Tenner also point out conclusively that the chief seat of epilepsy is located in the medulla oblongata; and Echeverria in his celebrated work on *Epilepsy*, very pertinently remarks on page 191:

To class phthisis among the diseases which are capable of super-inducing epilepsy in the offspring, may at first sight appear an altogether untenable opinion. However, a little consideration will manifest that it is perfectly consistent with the tendency of lesions of the medulla oblongata to induce pulmonary tuberculosis. . . . . Resting on the well-known experiments of Schiff who generated pulmonary tuberculosis, by dividing the ganglia of the pneumogastric in rabbits, Van der Kolk very significantly observes: 'Since, as experience has taught me, we so often see pulmonary consumption occur in families, some members of which are affected with insanity, so that I have often seen that children who were spared from insanity were the victims of phthisis, and that the two diseases frequently alternate with one another, or coexist, the question has often suggested itself to me, whether we might not admit the existence of a phthisis exentrica, namely, one

whose first cause is to be sought in an irritated condition of the medulla and the vagus, such as so frequently occur in phthisical subjects.'

On page 313 the same author (Echeverria) observes:

I have most closely investigated the relations of pulmonary tuberculosis and epilepsy, and undoubtedly the genesis of tubercles in the lungs is favored by the lesion in the medulla oblongata proper to epilepsy. I have traced the pulmonary trouble from its inception, and feel convinced that the association is more than a casual coincidence of both morbid conditions. I have been no less struck with the frequency of tubercles, or other pulmonary lesions, I have met with on post-mortem examination of epileptics.' And again on page 76 he says: 'In close relation to this subject I may say, that among my patients who died in or immediately upon an attack, all but two were those in whom the degeneration displayed itself more markedly in the vicinity and origin of the vagus; a result confirmatory of that of Van der Kolk and Kroon, who assert that those patients who did not bite the tongue had died in a fit. Brown-Séquard, quoting cases from Jobert de Lamballe, Stuart Cooper, and Rostan, as also Van der Kolk, have reported examples of disease of the Pons varolii and the medulla oblongata involving the vagus, and attended with pulmonary trouble. . . . . I have recently examined the pneumogastric and cervical sympathetic from a child who died with whooping-cough, and compared the changes they exhibit with those of the same organ from another epileptic child, both patients at Randall's Island Hospital, directed by my friend Dr. F. A. Castle, who kindly sent the specimens to

me. In the first case the connective fibres and nuclei and the capillaries, were very much increased in the ganglia and nerve, their condition being that of neuritis; in the latter instance, however, the hypergenesis of connective elements was not so high, and the cells and primitive fibres had undergone beside a fatty degeneration. In investigating the pathological changes of the sympathetic system and nerves connected with it, I have met with redness and enlargement of the pneumogastric in cases of pneumonia. . , . . In phthisis—and chiefly with children—there is a peculiar degeneration of the pneumogastric and cervical sympathetic ganglia.

Owing to the fact that the principal pathological lesion of epilepsy resides in the medulla oblongata, it is very easy to understand why this disease is so frequently associated with phthisis, if we accept that degeneration of the vagi, the nuclei and roots of which originate in this same tract of nerve tissue, form the underlying basis of the latter disease. Indeed, both Van der Kolk and Echeverria distinguish two forms of epilepsy in this respect: One in which the hypoglossus is affected and the tongue is bitten; and the other in which the vagi is involved and the tongue is not bitten, but in which there is greater danger of sudden death and of pulmonary complication.

We must look, therefore, more particularly among those epileptics in whom the vagi are diseased for the occurrence of pulmonary disease, and I have taken the liberty of tabulating twenty-four such examples from Dr. Echeverria's work:

NO.	AGE.	SEX.	DISEASE.	POST-MORTEM LUNG ; CONDITION.	POST-MORTEM VAGUS CONDITION.	PAGE OF DR ECHEVERRIA'S WORK.
115	Adult	F.	Alcoholism and epilepsy.	Pneumonia.	Medulla degenerated.	52
116	54	M.	Alcoholism and epilepsy.	Phthisis.	Medulla degenerated.	56
117	26	F.	Syphilitic epilepsy.	Phthisis.	Medulla degenerated.	72
118	20	M.	Epilepsy.	Phthisis.	Medulla degenerated.	84
119	3	M	Epilepsy	Pneumonia.	Medulla degenerated.	101
120	22	F.	Epilepsy.	Phthisis.	Nuclei of vagi and medulla degenerated.	103
121	24	M.	Epilepsy.	Phthisis.	Vagus and medulla degenerated.	105
122	35	M.	Epilepsy.	Double pneumouia.	Vagus and medulla diseased.	101
123	22	F.	Epilepsy.	Phthisis.	Vagus roots and medulla diseased.	103
124	24	M.	Epilepsy.	Phthisis.	Vagi and medulla diseased.	105
125	54	F.	Alcoholism and epilepsy.	Phthisis.	Medulla degenerated.	138
126	40	F.	Epilepsy.	Pneumonia.	Softening of vagi nuclei.	138
127	37	F.	Syphilis and epilepsy.	Phthisis.	Nuclei of vagus and medulla degenerated.	138
128	26	F.	Syphilis and epilepsy.	Phthisis.	Medulla and roots of vagi degenerated.	138
129	20	F.	Epilepsy.	Phthisis.	Amyloid degeneration of medulla	138
130	3	M.	Teething. Epilepsy.	Pneumonia.	Vagi, medulla and sympathetic degenerated	138
131	22	F.	Epilepsy.	Phthisis.	Vagi, hypoglossus and medulla degenerated.	138
132	24	M.	Epilepsy with dysphagia.	Phthisis.	Root of vagus and hypoglossus diseased.	143
133	16	M.	Epilepsy.	Phthisis.	Congestion around vagus.	143
134	31	F.	Alcoholism and epilepsy.	Pneumonia.	Vagus and sympathetic degenerated.	143
135	53	M.	Syphilis and epilepsy.	Phthisis.	Medulla degenerated	143
136	43	M.	Epilepsy.	Pneumonia.	Medulla degenerated.	143
137	68	F.	Epilepsy.	Phthisis.	Vagi and medulla degenerated.	143
138	18	M.	Epilepsy.	Phthisis.	Medulla degenerated.	143
139	40	F.	Epilepsy.	Phthisis.	Left vagus compressed by tumor in medulla.	143

CHAPTER VII.

RELATION BETWEEN DIABETES AND PULMONARY CONSUMPTION.

The statement is occasionally made that diabetes and tuberculosis rarely, if ever, co-exist. This has latterly been advocated by those to whom the bacillus is the centre around which all other ideas concerning pulmonary consumption revolve. That this belief does not rest on a sure foundation, can readily be ascertained by glancing at the facts. Dr. Copland (*Dictionary of Medicine*, vol. i, p. 508) states that he scarcely ever met a case that was entirely uncomplicated with pulmonic symptoms. Bouchardat (*Etiologie de la Tuberculisation pulmonaire*, in the supplement to *L'Annuaire de Thérapeutique* for 1861, p. 4) says that in nineteen *post-mortem* examinations of diabetic patients he found tubercles in every instance. In a discussion on the Morbid Anatomy of Diabetes, before the London Pathological Society (Trans. London Path. Soc., vol. 34, p. 336) Dr. Findlay said that he and his colleague, Dr. Coupland, had searched the *post-mortem* records of the Middlesex Hospital for a period of twenty-six years, and found particulars concerning twenty cases of diabetes, all of which except two had marked pulmonary and pleuritic lesions. Eleven of these were phthisical.

It may be further stated that these phthisical

lesions were often obviously of such old standing as almost to compel the conclusion that the diabetes arose in the subjects of tuberculosis, and not that the latter was secondary to the former (p. 53).

In the same discussion (p 355), Dr. Stephen Mackenzie reports thirty-seven cases of diabetes from the death records of the London Hospital during a period of nine years (from 1874 to 1883), twenty of which gave evidence of pulmonary phthisis. Dr. Mackenzie said :

From the above table it will be apparent that in many cases there were other conditions present, besides the diabetes, that may have had an influence in bringing about the death of the patient. In some instances it is difficult to determine the share such conditions took in causing, or being caused by, diabetes. Some, as phthisis pulmonalis, and renalis, and gangrene of the foot, are no doubt the outcome of diabetes. . . . . From a study of this series of fatal diabetic cases, two facts stand out prominently: The tendency of this disease to bring about phthisis; and to terminate in coma.

As to the dependence of the phthisis on the diabetes, I think no doubt can be well maintained. I know of no one other general disease which is so frequently succeeded or complicated by phthisis It would seem, indeed, that the natural mode of termination of diabetes is by inducing phthisis, but that in a large proportion of cases this stage is only partly reached, or not reached at all, owing to the patient being prematurely cut off by coma.

Dr. Frederic Taylor (p. 371) contributed fifty-

three cases of diabetes which occurred in Guy's Hospital during a period of nine years, fourteen of which were phthisical, and one pneumonic.

Diabetes may be classed among those diseases which depend on an error in the chemical changes of the body; yet, primarily, it is due to a disorder of the nerves which preside over these changes. Just as there are special nerves of motion, of sensation, and of heat regulation, so there are nerves which have special control over the trophic changes of the body. As a nutritive agent, sugar plays an essential rôle in the bodily economy. It is manufactured by the liver out of glycogen, and this organ also has the power of changing sugar back into glycogen again. This function is not, as is commonly supposed, exclusively confined to the liver, for the muscles, in all probability, have the same power, together with that of changing sugar into lactic acid and glycerin (Brunton).

Now, it has been well demonstrated that puncture of the fourth ventricle, irritation of the vagi roots, an injury to the medulla oblongata, or the administration of chloroform, curare, strychnine, interfere with this glycogenic metabolism, inasmuch as they render the body unable to transform its ingested sugar into working force. Hence this substance remains in the blood, and is secreted by the kidneys. The nerve centre which presides specially over this change can, therefore, be located in the

region of the medulla oblongata, while a more general power of the same kind is, in a measure, also shared by the peripheral nerves – hence, there may be two forms of diabetes, one dependent on a central and the other on a peripheral nerve lesion. We can readily understand, then, that the reason why pulmonary consumption and diabetes are so often associated is, because they both depend on the same fundamental nerve disorder. The following histories are typical examples of this:

Case 140.—Dr. A. Weichselbaum (*Diabetes mellitus bei multiple Shlerose des Gehirn u. Rückenmark.* Wiener Med. Wochenschrift, 1881, S. 914): Male, 34 years old, was received May 14th, 1880, and reports that he has been weak and sickly for two years. Had increased secretion of urine for six months. Passes from 2,500 to 9,600 c. c. of urine, which contains 3.4 p. c. sugar. Died October 31st of the same year. Section: Chronic tuberculosis of both lungs, with cavities. Sclerosis of the vagus and glosso-pharyngeal nucleus.

Case 141.—De Jonge (*Tumor der Medulla oblongata; Diabetes Mellitus.* Arch. f. Psych. xiii. S. 658): Male, aged 37, was received with symptoms of pulmonary tuberculosis and anasarca. He was markedly emaciated, had increased thirst, and on investigation it was found that his urine contained a trace of albumin and about 6 p. c. of sugar. He gradually became worse, and died shortly after his reception. Section showed pulmonary tuberculosis, and a tumor about the size of a bean reaching from the left olivary body to the first cervical nerve, and pressing on the parts beneath.

CHAPTER VIII.

RELATION BETWEEN BERI-BERI AND CONSUMPTION.

In Japan occurs an endemic peripheral nerve disease which is called Kak-ke (disease of the legs) by the natives, although it is similar to an affection of other countries known as beri-beri. This disease, as described by Dr. Scheube (*Deutsches Archiv. für Klin. Med.*, Bd. xxxi. S. 141, 307; Bd. 32, S. 83; also Virchow's *Archiv.*, Bd. 95, S. 146), to whom I am much indebted for most of my information, is not contagious, although infectious, and in nature is allied to intermittent fever, and is most prevalent among the Japanese in large cities. Those who follow indoor sedentary occupations, like teachers, students, priests, clerks, merchants, etc., are most liable to it. Among the predisposing causes are taking cold, getting wet, exhaustion of body and mind, emotional excitement, excesses, etc.

The disease begins to manifest itself by a weakness and heaviness in the lower extremities, which is often preceded by fever, and catarrh of the bronchi, stomach and intestinal canal. These symptoms are followed by anæsthesia and pain, as well as by œdema of the legs, by cardiac palpitation, dyspnœa, gastric oppression, indigestion, paralysis, etc. The paralysis generally confines itself to the lower extremities, al-

though sometimes it extends to the face, tongue, pharynx and larynx. Effusion into the large serous cavities frequently takes place, which is associated with diminished urinary secretion.

Kak-ke frequently develops in connection with other diseases, notably, typhus, cholera, intermittent fever, acute articular rheumatism, pleurisy, syphilis, pulmonary and laryngeal tuberculosis, sciatica, and after operations. Dr. Scheube says (p. 160) "that phthisical patients contract kak-ke, or that in the course of kak-ke the patient becomes phthisical, I have observed not only once but in a whole series of cases," and Prof. Baelz says that, according to his experience in Tokio, phthisis pulmonalis is the most frequent complication of kak-ke.

It is said that a variety of micro-organisms is found in the blood of those suffering from this disease, which if cultivated and introduced into the circulation of animals is capable of calling forth a characteristic multiple neuritis; although this is denied by Dr. E. van Dieren (*Schmidt's Jahrbücher*, Bd. 220, S. 99).

Dr. Scheube * made a careful examination of the bodies of twenty beri-beri cases and found effusion in the large serous cavities, anasarca degeneration of the skeleton muscles, dilatation, hypertrophy and fatty disease of the heart, as well as pulmonary em-

* Virchow's Archiv. Bd. 95, S. 146.

physema and tuberculosis. A microscopical investigation of the nervous system showed that the brain and spinal cord were generally healthy, but that such nerves as the crural, tibial, peroneus, phrenic, the vagus and its branches were generally diseased. Dr. Scheube further says that he is very much disposed to attribute the disturbance of the lungs and heart to disease of the vagus.

The following history is an illustrative example of this disease when it is associated with pulmonary phthisis:

Case 142.—Male, aged 20, seen for the first time on the second of May, 1879, and with the exception of a slight attack of kak-ke in August, 1878, was well until March 26th, 1879, at which time he suffered from a severe headache. One week after this he became feverish and thirsty. Middle of April he became afflicted with a gradually increasing weakness of the lower extremities. Simultaneously there were pain on pressure, and diminution of sensibility in these parts, as well as in the hands, but his appetite was good. On the first day of May he had a temperature of 39.0°, respirations 24–36, pulse 100–120. Galvanization of the spinal cord was ordered. Status praesens May 14th. Weak constitution, anæmic and thin, no œdema. There was less respiratory motion in left than in the right side of chest. Dullness in upper part of lung on same side, as well as crepitant râles posteriorily. Diffuse pulsation in cardiac area, and the dullness here extended to the middle of the sternum. The sitting position accelerated the pulse, the appetite was somewhat impaired, the tongue coated, and there were traces of albumin in the urine.

On June 20th, the lung affection was decidedly aggravated. The left clavicular region gave a dull tympanitic percussion sound which extended to the fourth intercostal space. Fever and diarrhœa set in, he became markedly emaciated, and died in collapse on the 29th of July.

This disease is regarded by Dr. Scheube, from whose work the above history is copied, and all others who have investigated it, as a multiple neuritis. It is believed to be caused by a specific micro-organisms by some, and by others it is ascribed to the influence of food, heredity, climatic conditions, etc. It is not contagious, according to the opinion of Dr. Scheube and of others who have had abundant opportunities of studying it in its native habitat.

CHAPTER IX.

RELATION BETWEEN LEPROSY AND CONSUMPTION.

Hillis, in his work on *Leprosy in British Guiana*, says on page 39, that "lung complications occurred in seventeen per cent. of his cases of tuberculated leprosy; and with regard to phthisis as a complication it would be interesting to study how far, if at all, tuberculosis in general may be related to lepra. May the one disease be, as it were, lit up by the other? I confess I am not in a position to discuss this matter, and I can only state that in the cases referred to in the table, it is believed there was a pre-disposition to phthisis. The post-mortem appearance of some of the cases differed in no way from those met with in ordinary fatal cases of consumption, except that they were subjects with advanced tuberculated lepra."

In his Lectures on Leprosy, Dr. Bidenkap says, that "lepers are frequently attacked by common tuberculosis which then becomes the cause of death. The relation is remarkable and not easy to explain, if we, as is universally believed, consider tuberculosis a specific infectious disease."

From the experience of these authors it is quite evident that pulmonary consumption is a frequent concomitant of leprosy, and in order to bring out the parallelism between these two diseases more fully I

propose to give a brief general survey of the symptoms and pathology of the latter, in which I shall endeavor to follow their teachings.

Leprosy is regarded as a cutaneous disease, though its ravages are by no means confined to the tissues of this structure. It is usually divided into the anæsthetic and tubercular forms, yet both forms are merely different stages of the same process varying only in the intensity of certain manifestations, and frequently become mixed in the same individual. The premonitory symptoms of both forms are general *malaise*, debility, mental depression and drowsiness, often associated with rheumatic-like pains in the extremities, hyperæsthesia, chills and fever. These are followed by pains along the course of the peripheral nerves, numbness of the skin, maculæ, bullæ, muscular atrophy, and later by ulceration and necrosis of the skin, mucous membranes, subcutaneous connective tissue, periosteum, bones, and of some of the internal organs.

The principal differences in the two varieties are in the onset of the attack and in the mode of eruption. In the *anæsthetic* variety, after the above described symptoms may have lasted for nearly a year, spots of a pale yellow color, of the size of an inch or two in diameter, and not raised above the surface, are noticed. These spots usually correspond to the distribution of a nerve, and evidence of nerve disease exists sometimes before the eruption appears. Then

the nerve swells and can often be felt through the skin. The most frequent sites of these eruptions are on the back, shoulders, posterior parts of the arms, nates, thighs, around the knees, elbows, and the face. In from two to three years after the beginning of the disease the edges of these spots become raised, and then enlarge. The hands and feet now become devoid of feeling, bullæ, and ulcers which are conical in shape, form with blackened edges and exude a thin unhealthy pus. The ulcer's apex extends down to the bone, which necroses, and can eventually be removed with the forceps. These ulcers are very apt to occur on the sole of the feet of people who walk barefooted. When the bone is near the surface as in the ankles and wrists, or in the hands and feet, the destructive process reaches the periosteum, the bone is laid bare, caries begins, and a large portion of the affected bone may be destroyed. Sometimes the ulceration enters articular cavities, and gives rise to inflammation, continued suppuration and to spontaneous amputation. The phalanges and then the meta-carpal and meta-tarsal bones are most liable. This form may last from eighteen to twenty years.

The *tuberculated* variety is characterized by an eruption of tubercles, and after the above described premonitory stage, erythematous, reddish-brown patches, in size from a shilling to asaucer, appear on the skin. After the eruption subsides, tubercles of the size of a pea form on the site of these patches. There is now

also diminished cutaneous sensibility in the patch, although before this there is hyperæsthesia. These patches become elevated and œdematous. Successive crops of tubercles break out, which are preceded by pain and aching in the limbs, and accompanied by febrile attacks. After a tubercular eruption, the fever and *malaise* usually disappear, but often one eruption with fever succeeds another for a very long time before a remission appears. The tubercles soften, ulcerate, and abscesses form in the skin, which destroy the deeper tissues—bone and cartilage—in a manner similar to those which occur in the anæsthetic variety. The mucous membrane of the throat, mouth, and nose is attacked, and eventually that of the larynx. The tubercular infiltration in these parts is reddish, and develops slowly. The vocal cords usually have a tuberculated and uneven appearance, and the voice is hoarse.

The essential element in leprosy is a lesion of the peripheral nerves. This is universally admitted, so far as the anæsthetic variety is concerned, but in the tuberculated form it is believed by some that the nerves are secondarily involved. By reason of the facts that no sharp line can be drawn between the two varieties, and that they often intermix in the same patient, it seems to be inconsistent to deny to one what is accorded to the other, so far as their primary lesion is concerned. Danielssen, in his pathological investigations, found that the smaller cutaneous

branches of nerves running to the affected spots in the skin were red and swollen, especially the neurilemma. Later in the disease these nerves had a more brownish color, especially the perineurium, which became tumefied and compresed the nerve fibres. In old cases swelling of the nerves could be traced as far as the brachial plexus and the sciatic. At last the nerves lost their swelling, became yellowish and atrophied, and the axis cylinder disappeared. Virchow confirms this view of the pathology of the disease. Danielssen believes that the inflammation in the sheath of the nerve gives rise to the deposit, while Hillis believes the reverse, and Charcot thinks it a neuritis. Charcot says (Lectures on Diseases of the Nervous System. Trans. by Dr. Sigerson. *New Syd. Soc.*, p. 26), "it is above all in anæsthetic lepra that we encounter in their full development the trophic disorders which we have studied in connection with traumatic nerve lesions. The first of which includes eruptions of various forms, but chiefly those characterized by vesicles and bullæ; the second pemphigoid eruptions. . . . . Here we see the pemphigoid bullæ developing with great rapidity, and reappearing from time to time, on different parts of the tegumentary system supplied by the wounded nerve." Brown-Séquard's experiments on Guinea-pigs, etc., showed that ulceration of the toes, and loss of nails, etc., took place on section of the sciatic nerves. Bacilli are found in the disease, and the tubercles are genuine tubercles with giant cells.

It has already been stated that pulmonary consumption is frequently associated with leprosy. The following is a typical case of this kind:

Case 143:—Male, 25 years old, was brought to Rigs Hospital in Christiana, October, 1880, who on admission suffered from fully-developed tuberous leprosy, with extensive deposits, especially on the face and extremities. At the same time there was a swelling of the ulnar nerves and anæsthetic spots on the forearm and hands, together with some muscular atrophy of the hands. His voice was hoarse, the vocal cords were swollen, the mucous membranes reddened, with leprous deposits in the throat and on the Schneiderian membrane. He had frequent attacks of pyrexia. During these attacks there often appeared eruptions of reddish infiltrations the size of a walnut on the face and on the extremities, some of which suppurated, while other tubers softened. At the same time the nerve affection increased, more extensive anæsthesia became manifest on the upper and lower extremities, together with soreness and swelling of the large nerve trunk. The leprous deposits in the throat and nose softened, and the point of the nose sank in. He also suffered from severe neuralgic pain, and emaciated greatly. Added to this was a bronchial catarrh with frequent exacerbations, and the usual symptoms indicating consolidation of the apices of the lungs, as well as recurring slight pleuritis. Then ensued gastric disturbances, irregular purging, with frequent attacks of diarrhœa and pain in the abdomen. Towards the end of 1882 all the leprous spots began to disappear. Even the recent infiltrations retrogressed partly by suppuration, and when he died, in June, 1883, all the

leprous tubercles and infiltrations had totally disappeared, even those on the mucous membranes. On post mortem investigation, common tuberculosis was found to be excessively developed in both lungs in the form of recent outbreaks and old caverns. Miliary tuberculosis was also present in the peritoneum; tubercular ulcers in the intestinal canal, and amyloid degeneration of the spleen and portion of the kidneys. Altogether there were but few signs of leprosy on his body. The peripheral nerves, especially the ulnar, were swollen. The tuberculosis in this case caused the leprous process to recede, and the patient died of tuberculosis instead of leprosy. This is the frequent termination of the latter disease. (Bidenkap, p. 27).

So far as the ætiology of leprosy is concerned, it may be stated that all sides concede that intermarriage must account for a certain number of cases. Heredity is all but universally acknowledged. Danielssen and Boeck insist that this is the chief cause of the propagation of the disease in Norway. Of 1,564 individual lepers, the following number and proportion of blood relations were likewise affected:

DIRECT LINE.

Father	684	43.8 per cent.
Mother	197	12.6 "
Grandparents	53	3.4 "

COLLATERAL LINE.

Uncles, aunts, etc,	226	14.5 per cent.

CO-EQUAL LINE.

Brothers and sisters	334	4.3 per cent

When the father has tuberculated leprosy, and the mother anæsthetic leprosy, the offspring will most frequently be affected with mixed leprosy. In the West Indies, children of lepers are often scattered abroad previous to the age at which the nerve affection usually appears. These grow up, become lepers, beget children, and all chance of obtaining a family history is lost. In these cases there is no assignable cause to a disease which in all probability has been hereditarily acquired.

Common opinion holds leprosy to be a *contagious* disease. But in 1867, a Committee of the College of Physicians of London prepared a series of questions in regard to its contagiousness, which were sent abroad; and in its report this committee states that "the almost unanimous conviction of most experienced observers in different parts of the world is quite opposed to the belief that leprosy is contagious, or communicable by proximity or contact with the diseased."

The *treatment* of leprosy is chiefly constitutional. Specific medication, with a view of annihilating the bacillus, which by some is supposed to be the cause of the disease, has proved a failure. Nerve-stretching, and tonic treatment, have been attended with success. (*Lancet*, Feb. 26th, 1881.)

CHAPTER X.

RELATION BETWEEN LUPUS, PELLAGRA, MERCURY, LEAD, ETC., AND CONSUMPTION.

Lupus has long been regarded as a tubercular disease, and now, since the tubercle bacillus is said to be found in it, this is almost positively proved. Whatever else may be said, it is quite certain that lupus is closely affiliated with tuberculous disease of the lungs. Of thirty-eight cases of lupus seen by Besniers (*le lupus et sou traitement.* Ann. de Dermat. et Syph., 1883, page 383), eight were phthisical. Block (*Klin. Beiträge zur Ætiologie u. Pathogenesis des Lupus vulgaris*—Vierteljahrschrift f. Dermat. u. Syph., 1886, s. 238) states that out of one hundred and forty-four lupus patients, there were one hundred and fourteen who suffered from tuberculosis of other organs than the skin. There was a marked hereditary history in one hundred and six of these cases. Of nine deaths among them, eight were caused by pulmonary tuberculosis. Of seven deaths among lupus patients, reported by Sachs (*Beiträge zur Statistik d. Lupus*—Vierteljahrschr. f. Dermat. u. Syph., 1886, page 241), six were due to pulmonary phthisis. Bender (*Ueber die Beziehungen des Lupus vulgaris zur Tuberculose*—Deutsche med. Wochenschr., 1886, No. 23 u. 24) contributes one hundred and fifty-nine cases, ninety-nine of which were tuberculous.

Although the pathology of lupus is not well made out, yet it is of great interest to find that the nerves of the skin are seriously involved in the disease. Thus Dr. Morison (*Histologische Untersuchungen über Lupus*—Vierteljahrschrift für Dermatologie u. Syphilis, Bd. 13, 1886, S. 623) found that the nerves which supply the sweat and sebaceous glands were markedly degenerated and infiltrated. This was not only the case with the perineurium, but the nerve-fibres themselves were seriously involved; they were nodular, uneven, and swollen. Dr. H. Leloir, a most distinguished French authority on skin diseases, says in his *Recherches cliniques et anatomo-pàthologiques sur les affections cutanées d'origine nerveuse*, p. 120, that lupus vulgaris and lupus erythematosus are primarily dependent on diseases of the peripheral nerves.

Another disease which in many respects bears a close analogy to beri-beri and leprosy is pellagra—a disease common in Italy, Spain and in the south of France, and believed to be due to the exclusive consumption of maize in a state of deterioration. In Italy alone there are probably one hundred thousand peasants suffering from it at the present time. It is characterized by symptoms of diminished strength by an erythematous eruption, and by marked disturbance of the nervous system, such as pain in the head and spine which radiates to the arms, legs, and abdomen. The first symptoms are stupefaction, giddiness with a sense of traction from

behind, and general debility. The skin assumes an earthy color, shows an erythematous discoloration with desquamation of the cuticle, after which come rough nodosities, vesicles and puckerings. The patient becomes more feeble, emaciates greatly, totters with knees semi-flexed, and suffer from labored breathing which sometimes approaches absolute apnœa, The pulse is rapid, and the impulse of the heart becomes weaker and diminishes in bulk as the organ shrinks. There is often mental alienation, and more than one half of the deaths among these people are caused by pulmonary consumption. (*Lancet*, 1880, Vol. I, p. 148.)

In the present condition of pathology we do not know whether the same nervous link connects lupus and pellagra with pulmonary consumption as that which has been shown to exist between alcoholism, syphilis, diabetes, beri-beri and leprosy and the latter disease. All that can be safely said is that inference points in that direction. These diseases were not introduced here in the belief that they will greatly strengthen the direct argument that consumption is a nervous disease, but merely to show that both are probably neuroses, and that further investigation may indicate that they bear the same relation to the disease in question as those do which have received more thorough consideration from the hands of pathologists.

Reference must also be made here to the fact

that certain metallic agents like mercury, lead, arsenic, etc., likewise have the power of originating phthisis, as well as other forms of pulmonary disease. The vapor of mercury when inhaled for a long time, as in the case of those who are employed in mining the metal, or who come in contact with it in manufactories, occasions tremor, unsteadiness of the hands and arms, dizziness, loss of memory, convulsions, epilepsy, delirium, paralysis and death, which, according to Professor Binz, is brought about by paralysis of the cardiac and respiratory centres. Its physiological action therefore demonstrates its specific affinity for the nervous system. The workmen of Paris who were engaged in the manufacture of looking-glasses in 1821, "could not remain at the trade above eight or twelve years. When necessity compelled them to persevere too long, their faces became pale, with an expression of intoxication, their intelligence and memory gradually failed, they fell into a kind of idiocy, and after lingering in this state for some years, they died of consumption, or were struck with apoplexy. Dr. Sauderet, in 1847, reports that the trade of water-gilding at Besanson, where it was extensively carried on, was most injurious to health, the mortality among the workmen being enormous, and due chiefly to phthisis."* Professor Binz† relates a

*Art. Metallic Tremor. Reymond's System of Med. Vol. 2, p. 357.

† Vorlesungen über Pharmacologie, Seite 619.

very interesting incident which took place on board a man-of-war, in which mercury, constituting a part of the cargo, poisoned all the sailors who were exposed to its fumes. He says that those who had any predisposition to pulmonary disease suffered very much from it. Five contracted phthisis in a very short time, three of whom died.

Dr. Hussmaul (Ueber Constitutionellen Mercurialismus) states that about 50 per cent. of those engaged in mercury manufacture, die of pulmonary consumption.

Lead is also well known as a nerve poison, and many practitioners are familiar with a form of phthisis which occurs among those who suffer from the injurious effects of lead. The following histories give two examples of this kind.

Case 144.—(Dr. Carmuset. *Ein Fall von Chronischer Bleivergiftung.* Clbt. für Nervenheilkunde, Bd. 7, 1884, p. 69.) Male, aged 48, painter, suffered from lead colic, tremor of tongue and hands, weakness of the muscles and unsteady gait. Tuberculosis of the lungs supervened, which finally caused his death, after which it was found that his brain contained thirty-six mgr. of lead.

Case 145.—(Dr. Oppenheim. *Zur Pathologischen Anatomie der Bleilähmung.* Archiv. f. Psych., xvi, S. 376. Clbt. f. Nervenheilkunde, Band 8, 1885, S. 533.) Male, aged 33, type founder, who had for twenty years been working in lead and in lead dust. He frequently suffered from colic, and in 1879 became subject to weakness in the lower as well as in the

upper extremities. Examination at this time showed typical saturnine extensor paralysis, and a trophic paralysis in the lower extremities. He improved under electrical treatment. In 1884 he became worse. Stomatitis and brain disturbance set in. The upper extremities became fully paralyzed, and all the changes of chronic lead poisoning manifested themselves. Death occurred four days after admission. Section: Catarrhal pneumonia, granular atrophy of the kidney, and hypertrophy of the heart. Marked degeneration of most of the muscles of the lower extremities and also of the post-tibial nerves. The anterior grey horns and probably the whole spine was diseased. Nothing is said concerning the condition of the vagi.

Evidence can also be adduced to show that arsenic, brass, and other substances are capable of producing pulmonary consumption.

CHAPTER XI.

THE SYMPTOMS OF CONSUMPTION STUDIED FROM THE STANDPOINT OF THEIR NERVOUS ORIGIN.

Apparently the symptoms common to pulmonary consumption stand in a disconnected relation to each other. That this is not true, however, and that a complete harmony exists in regard to their origin, course and termination, will be seen from the following considerations.

Fatigue and Exhaustion.—Probably one of the earliest manifestations of the advent of pulmonary consumption is a tendency to premature bodily fatigue. While it is true that members of phthisical families are, as a rule, vivacious, intellectual, and indeed sometimes precocious, it is also true that their nervous system is in an unstable equilibrium, and is, therefore, easily disturbed, and they further lack that reserve power of physical endurance which is present in individuals not so predisposed. This want of energy is generally among other things attributed to muscular weakness. That the muscular system participates in this debility is beyond doubt, but there is good reason for suspecting that the principle difficulty is not located in these structures, but in their supplying nerves. Another reason frequently given for the exhausted state of the muscular system is the impair-

ment of breathing surface which occurs in the course of this disease. When we reflect, however, that the amount of breathing surface normally far exceeds the oxygen demands of the body, it is hardly possible for this to be a serious obstacle to the proper oxygenation of the blood. If this defective power is absent at the beginning of the disease, it seldom fails to appear throughout its course, and it often becomes one of the most annoying symptoms. There are some cases of consumption which apparently do well, so far as cough, expectoration, and appetite are concerned, but are unable to perform the least physical exertion. In spite of the good signs mentioned, they are on a constant decline. Walking, or going up a flight of stairs, saps their strength at once. Such patients I have known to suffer from violent vertigo, tremors, numbness in the hands and feet, as well as from the most agonizing pains in the back and in the extremities, clearly showing that there was present either a neuritis or a neuralgia.

That degeneration of the peripheral nerves is frequently associated with pulmonary consumption, has already been shown by the general evidence, but in addition to this we have the special investigation of Dr. Jappa.* His work comprises the histological

*Zur Frage über die Veränderungen der peripherischen Nerven bei Schwindsucht. Dissertation. St. Petersburg. 1888. Abstracted in Neurologisches Centralblatt. Bd. 7. 1888. Seite 425.

examinations of the peripheral nerves in the bodies of fifteen persons who died of pulmonary tuberculosis. The nerves which came under his examination were the sciatic, post-tibial, internal plantar, crural, internal saphenous, superficial peroneus, median, ulnar, radial, cutaneous median, and radial interosseus. In every examination most of the twelve excised peripheral nerves showed marked degenerative changes. The changes were located in the axis cylinder, perineurium, as well as in the sheath of Schwann, and the author says that they should be regarded as parenchymatous in character. The intensity of the morbid changes was greater in the peripheral endings than in the trunks of the nerves. The spinal cord was examined in twelve cases and found to be normal. Now, in all these cases the author says there were no manifestations of nervous symptoms during life other than those of some undefined neuralgias and muscle pains, general hyperæsthesia, and which are believed develop in the high fever and inanition of phthisical patients.

Loss of Appetite.—The dyspepsia of pulmonary consumption has been attributed to a great variety of causes, and it constitutes one of the most annoying features in the treatment of this disease. The persistent failure to eat is so marked that one is often tempted to ask whether the real seat of the trouble does not reside in the stomach instead of in the respiratory organs! That the real difficulty lies

deeper than simply a defective gastric or pancreatic secretion, and is in close affiliation with the nervous system, may possibly be inferred from the powerful influence which emotional and peripheral impressions possess over the digestive power of the consumptive's stomach. Nothing proves more deleterious to his appetite than fright, disappointment, pain, etc.; and nothing often makes him eat better than when he is humored, coaxed, and encouraged.

When we come to consider that the lungs, the liver, and the small intestines, are largely supplied by the pneumogastric nerves, it requires no effort of the imagination to comprehend how disease in the former organs may readily disturb the process of digestion in a direct or reflex manner. The experiments of Bernard and others show that on section or irritation of the pneumogastric nerves the walls of the stomach cease to contract, the gastric mucous membrane becomes pale and flaccid, the secretion of gastric juice is arrested, the secreting function of the liver is deranged, and the small intestines are paralyzed. When these experimental facts, which demonstrate that the complex function of digestion is wonderfully dominated by the nervous system, are taken in connection with the *post mortem* changes which have been found in the nervous structures of individuals whose deaths were caused by phthisis, especially the alterations in the pneumogastric nerves described by J. Henle and others, there remains but

little doubt that the neurotic element plays a most prominent rôle in the process of phthisical dyspepsia.

The following cases show very distinctly that vagus disintegration disturbs hunger and thirst:

Case 146.—Longet (*Anatomie und Physiologie des Nervensystems*, 1849, Bd. II, S. 313) contributes an observation of Johnson, where in a case of softening of the medulla oblongata and compression of the root of the left vagus by an aneurismal enlargement of the left vertebral artery, complete lost of hunger and thirst occurred.

Case 147.—Brignardi (Longet, *supra*, Bd. II, S. 313) found in the obduction of a woman, who died of phthisis, and who had a voracious appetite during the latter part of her life, that both vagi were affected with red neuromatous swellings.

Case 148.—Swan (*Treatise on Diseases and Injuries of the Nerves*. London: 1834. Pp. 170) saw a patient who, in spite of perceptible integrity of the lungs, suffered from dyspnœa. Her appetite became voracious without being fully able to satisfy it, she vomited undigested food which had been eaten several hours before, and gradually emaciated and died. At the post-mortem examination, it was found that both vagi had atrophied. In this connection it may also be stated that vagotomy frequently brings on an unnatural appetite, or abolishes it altogether, in animals.

The dyspepsia of pulmonary consumption may, therefore, in common with many other symptoms, be regarded as one of the sign-posts which discloses the state of the nervous system. This induction tallies fairly well with that which is found in the practice of

every physician. It is always well understood that the prospects of a phthisical patient, who retains or recovers his appetite permanently, are brighter than if the opposite holds true. This is not alone due to the fact that he eats and assimilates more food-stuff, but because his innervation is fairly good, and because his vital forces are but little impaired.

Wasting.—The problem of nutrition has an important bearing on the pathology of pulmonary consumption. Wasting is one of the earliest symptoms which presents itself in the development of this disease, and while a great deal of this is undoubtedly directly due to a diminution in the quantity of food ingested, so common in this disease, no one will, I think, claim that the whole loss is due to this cause alone. There are many cases of incipient phthisis which, though they show no perceptible morbid changes in the lungs, and no apparent symptoms of dyspepsia, still undergo a gradual loss in flesh and strength; and there are others which, during the course of the disease, retain a tolerably good appetite, but become flabby and emaciate. From the general and special evidence already adduced, that serious functional and structural changes occur in the nervous system throughout this disease, and from the direct proof which will be further offered in support of the doctrine that the processes of waste and repair are largely influenced by the nervous system, I think it will become very obvious that the latter plays an essen-

tial part in the emaciation of pulmonary consumption.

The tendency of modern physiology is preëminently towards specialization This is particularly true of the researches which relate to the nervous system. The nerves and centres of motion and of sensation are already well demonstrated; the same may probably be said of the nerves and centres which preside over the production and dissipation of bodily heat; and, although their exact nature is still in doubt, there can be no question about the existence of nerves, be they spinal or ganglionic, which preside over the nutritive processes of the body. The existence of such nerves is well established by the following clinical data:* In his "Lectures on Nutrition," Sir James Paget relates the case of a patient in which the median nerve was compressed by the callus thrown out to repair a fractured radius. Ulceration of the thumb and middle fingers took place, which resisted all treatment until the wrist was so bound that the parts on the palmar aspect were relaxed and the pressure on the nerve removed. So long as this was done the ulcers became and remained well; if not, the ulceration returned.

Dr. John H. Packard records a very interesting

* Some of the instances which I give here are drawn from a paper entitled "The Influence of the Nerves on Nutrition," contributed to the Practitioner, vol. x, pp. 91, 138, by Dr. Henry Power.

case of trophic nerve lesion, in the *American Journal of the Medical Sciences*, April, 1870. A girl 11 years old was brought to him with a large splinter of wood under the ulnar side of her right thumb-nail, which had entered three days previously. Poultices had been applied and pus was beginning to form. He at once removed the splinter, and incised the thumb to afford a free exit to the pus. Relief followed, and the wound healed kindly, although the swelling was slow in subsiding. Three months after, the child was brought to him suffering under very grave general symptoms of nervous irritation. She had choreic movements of the whole body, of all the limbs, and of the jaw, the right half of her person being somewhat more affected than the left. She had lost flesh and strength, was peevish, irritable, and unable to fix her attention on anything. Her appetite was bad. Locally there was sensitiveness of the affected thumb, which she could not use in grasping, writing, or sewing. Iron, quinine, and arsenic were ordered as general remedies; the thumb was protected by an opium and belladonna plaster, and she was sent to the seaside. She improved some but the chorea continued as before, and on her return she again began to lose condition, and now there appeared one point of special sensitiveness at the ulnar edge of the thumb-nail. Exsection of the implicated nerve-filament relieved the child in a short time. In a few days she was steadier, she gained in flesh and strength, and in two months was perfectly well.

In the same number of the *American Journal of the Medical Sciences*, Dr. Harrison Allen reports the case of a cavalry captain who received a pistol wound in the left side of the neck, one inch above the clavicle, a little to the outer side of the sterno-cleido-mastoideus. On recovering after four hours' unconsciousness, there was found paralysis of both lower extremities, with intense burning pain in the left hand. The power of movement in the lower extremities was regained to such an extent that he could walk with crutches in five months, but the burning pain in the hand remained for ten months. This upper limb was numb, its nutrition was impaired. Three years from the date of the wound he died, and a particle of lead was found between the left brachial plexus and the corresponding subclavian artery, and the ball was lying on the side of the spinous process of the second dorsal vertebra. The spinal cord was congested, but free from lesion.

Messrs. Mitchell, Morehouse, and Keen, in their work on "Gunshot Wounds and other Injuries of Nerves," found the following trophic lesions due to nerve injuries: Atrophy of muscles, thickening of the cuticle, cracks and fissures of the skin, bed-sores, curved nails like talons, etc.

Lesions of the spine are frequently followed by trophic changes below the seat of injury. Charcot has collected a great many cases of this kind. Sir Benjamin Brodie observed sloughing of the heels

twenty-four hours after a lesion of the spine. Jeffreys mentions a case where a man fell and crushed the fourth dorsal vertebra, and on the fourth day an eschar made its appearance on the sacrum. Colling relates a case of dislocation of the seventh cervical on the first dorsal vertebra. Here also eschars formed on the sacrum on the eighth day, and on the heels on the fifteenth day.

The experimental evidence also points very strongly in the same direction. The most notable, and the only one of these examples to which I shall refer, is that of division of the trigeminus. Section of this nerve within the cranium is followed by a loss of sensation of that part of the face to which it is distributed, the cornea becomes cloudy, the nasal chambers are inflamed, and ulcers appear on the lips and gums.

Hoarseness and Aphonia.—Hoarseness often precedes as well as accompanies affections of the lungs, and especially is this the case in pulmonary consumption. In many, if not in most, instances this symptom is of a purely neurotic character, although it occasionally excites the suspicion that serious structural changes occur in tissues other than the throat. Since my attention has been drawn to the frequent implication of the nervous system in diseases of the respiratory organs, I have observed several cases of hoarseness, amounting to partial aphonia, dependent upon paralysis of one or of both vocal cords, which

on examination were found to be suffering simultaneously with an inflammatory consolidation of the posterior and lateral bases of both lungs, and which, together with the hoarseness, disappeared upon the administration of remedies which were almost entirely addressed to the nervous system and to the building up of the constitution. In all probability both of these affections, or symptoms of a single disorder, depended on defective pneumogastric innervation, since both of the involved areas are supplied by the same nerve-root. Sir Morell Mackenzie says that debility and hysteria are undoubtedly the most frequent causes of aphonia. This is common in the second and third stages of phthisis, and it is very often erroneously ascribed to structural changes, which, on examination, are found to be non-existent. In thirty-seven cases of phthisis, in which the voice was affected, he found that in eleven the affection was purely functional, in twelve there was thickening of the mucous membrane, and in fourteen there was congestion. He holds that in these cases of functional aphonia the nerve-force is feebly or imperfectly evolved, or it is not directed in the proper channel.*

The following is an admirable illustration of this condition:

Case 149.—Baumler (*Deutsches Archiv. für Kli-*

* "Use of the Laryngoscope in Diseases of the Throat," Morell Mackenzie, M.D. Second edition. Philadelphia, 1869.

nische Med Bd. 37, S. 231): Male, 61 years old, was admitted on the 15th of May, 1878, and during four months previous to this date he had been suffering from dyspnœa, hoarseness, profuse expectoration, cough, and stitches in his left side. At this time he had consolidation of both apices, emphysema, pleurisy between second and fourth ribs on left side, and signs of cardiac dilatation. Dyspnœa increased, cyanosis, a systolic mitral murmur, dropsy,and albuminuria supervened, and death took place on the 23d of the following June. During life a laryngoscopic examination showed a normal mucous membrane, and an almost complete immobility of the left vocal cord.

Section: Induration of both apices, pleurisy, peribronchial nodules in lower lobes as well as cylindrical bronchial enlargement. Heart was large and dilated. In the aortic region, the left recurrent laryngeal was found overgrown by an enlarged bronchial gland.

Case 150. – Krauss (*Centralblatt für Nervenheilkunde.* Bd. 9, S. 715): Male, aged 45, syphilitic, became afflicted with neuralgic pains at the age of thirty-five, which were followed by ataxia and difficult respiration—the latter being due to crico-arytænoid paralysis. Death caused by apnœa. Section: Slight degeneration in the sciatic nerve, marked degeneration in one vagus as well as in both recurrent laryngeal nerves.

Case 151.—Robert (*Gaz. des Hôp.*, 1853, p. 143), in tying the carotid, also included the vagus of that side. The patient at once began to scream that he would suffocate, became hoarse at once, and fainted. On loosening the ligature he fully recovered, but remained hoarse for six months.

Dyspnœa.—In speaking of the dyspnœa of pulmonary consumption, I do not refer to that form

which arises on account of extensive inflammation of the lungs, or of an accumulation of catarrhal material in the small bronchial tubes, and which act as an obstruction to the normal aëration of the blood, nor of that which is due to emphysema, or pseudo-asthma; but to that form which manifests itself as an oppression of, or a tightness in, the chest, and which is as much a neurosis as a genuine attack of asthma. According to my experience, this symptom is most prevalent in those cases of phthisis which suffer from the effects of excessive physical labor, from those of indulgence in alcohol or in other modes of dissipation. It is not so intense, nor are its limits so sharply drawn, as is the dyspnœa of asthma, but it seems to depend on the constitutional condition of the patient, being very much exaggerated when he is weak, and disappearing almost altogether when he is strong. I have seen such patients too debilitated to walk upstairs without panting, who, after a few days of well-directed medication, and that solely applied to the nervous system, became so much relieved that they were able to walk quite long distances without any well-marked dyspnœa. The very fact that this condition is susceptible of being changed through the agency of measures addressed only to the nervous system, is evidence that the whole disturbance is probably due to atony of the pneumogastric nerve.

Case 152.—(Wallis, *Hygiea*, xlvi, 9, S. 545). A male patient aged 33, who for a number of years had

been suffering with cough and hoarseness, was taken ill with pleurisy and bronchitis associated with fever. After the pleurisy disappeared he continued to cough and became short of breath, which grew into a strongly marked dyspnœa. There were dullness and the absence of respiratory sounds in the anterior portion of left lung. The dyspnœa increased, œdema set in, and he died. Section: Neuromatous swellings of the superficial nerves of the arms and legs, as well as of the deep seated nerves. Both vagi were hypertrophied. These nerves in the region of the thyroid gland were as large as an ordinary sciatic nerve, and at intervals had nodular swellings on them. Brain and spinal cord were anæmic but normal. The right lung was œdematous and partly consolidated, while the left was dry and had undergone fibroid degeneration.

Case 153.—Andral (cited by Longet., *supra*, p. 312) observed a persistent dyspnœa in a patient, without perceptible cause. Section showed that both vagi were surrounded and compressed by enlarged lymphatic glands.

Sweating.—It is quite obvious that, as a rule, perspiration is accompanied by dilatation of the cutaneous blood-vessels; but the experiments of Goltz, Luchsinger, Ott, and others, point out that this capillary expansion is but a passive factor in the production of sweat. These authors show that stimulation of certain nerves calls the sudoriparous glands into action independent of any vascular widening. Thus, in the dog and cat, increased sweating takes place when the sciatic nerve is stimulated, even though the aorta is divided or ligatured. These nerve-fibres are

probably regulated or co-ordinated by a centre or centres located in the spinal cord or brain, although the researches of Ott render it exceedingly likely that the sweat-glands may be excited through peripheral stimulation.* The profuse perspiration which accompanies the death agony, or violent emotional excitement, and many other pathological facts, likewise point out that the sweat-glands are dominated by the nervous system. Now, while sweating is not a specific symptom of pulmonary consumption, it is still one of its earliest and most persistent companions, and from the above premises it is quite certain that we are justified in believing that the exhausting cold and clammy sweats of this disease indicate profound disturbance of at least the peripheral portion of the nervous system.

Diarrhœa.—The persistent diarrhœa of pulmonary consumption is generally regarded as good proof of intestinal ulceration. While this may be true in a great many instances, I am quite certain, from a moderate amount of post-mortem experience, that diarrhœa may continue stubbornly for many months in this disease without showing the least evidence of intestinal ulceration after death. Indeed, I think it is a debatable question whether tubercular ulceration is ever the original cause of the diarrhœa, or whether

* "Sweat Centres. the Effect of Muscarine and Atropine on them," Dr. Isaac Ott, Journal of Physiology, vol. i, p. 193.

both the ulceration and the diarrhœa are the natural sequences of a common and more deeply-seated cause. Without venturing an opinion as to the nature of this cause, it must be admitted, on the score of common experience, that diarrhœa does occur both in children and in adults, which is attributable to nothing but disordered innervation of the bowels. The dominating influence of the nervous system over intestinal excretion is still further demonstrated by the experiments of Moreau, and of Brunton and Pye Smith, which show that, after destruction of the inferior ganglia of the solar plexus and the superior mesenteric, the intestinal canal fills with an abundant serous secretion. In view of these facts, are there not strong grounds for believing that the diarrhœa of pulmonary consumption may, in many of its phases, be due to a weakened and relaxed tone of the intestinal vaso-motor nerves?

Hæmoptysis.—The hæmoptysis to which reference is made here is that which occurs during the initial period of pulmonary consumption, and not that which is due to ulceration and which accompanies the later stage of the disease. During the former period either hæmoptysis or blood-spitting is rarely absent, and it usually marks the first step in the process of disintegration. Patients of this class come before you with the assurance that they are in perfect health, and to them this attack of blood-spitting is wholly unaccountable. On inquiry it will generally be found,

however, that they have been losing flesh for some time, have a poor appetite are employed in-doors, are overworked, and, perhaps, intemperate, have an active circulation, a florid complexion, and, above all, are of an excitable, nervous temperament. It may be pertinently asked: Why does pulmonary consumption make its overt beginning in a rupture of the lung capillaries? Why is the strong tendency in this disease towards a break in the continuity of the pulmonary circulation, while in acute pneumonia, where the blood-pressure is much greater, it is extremely exceptional to find more than a simple extravasation of blood in the air-cells? Inference leads one to suspect greater weakness in the walls of the blood-vessels in the former than in the latter disease, and we shall see that this suspicion is well borne out by the facts. The state of denutrition, which is the great bane of pulmonary consumption, and which, as we have already learned, is probably in a great measure brought about through the instrumentality of the nervous system, implicates the walls of the lung capillaries, as well as the other textures, and these in consequence give way to the slightest excess of pressure. That defective nutrition of the capillary walls plays a most important part in this disease is well supported by the testimony of Dr. Anderson, given in his excellent little work on "Phosphates in Nutrition." He says (page 125), "In all cases of death from consumption or analogous diseases that I have yet had

chances of examining, this deficiency (of inorganic matter) has been well-marked, and leads me to look upon it as an invariable characteristic of the wasting organic diseases, and if the symptoms, points of analogy, and general pathology of these diseases be considered, it will be seen that the theory of capillary inefficiency can be made to explain many of the phenomena of these diseases."

If we accept the theory of the close relationship between disordered innervation and pulmonary consumption, which I have endeavored to trace thus far, it is quite probable that the influence of the trophic nerves have something to do with the defective nutrition of the blood capillaries and hæmoptysis, which are referred to by Dr. Anderson in the work above quoted. Certain it is, according to the experiments of Brown-Séquard, that injury to the base of the brain in animals produces hæmorrhage in the lungs. There is probably very little doubt, then, that the weakened walls of the lung capillaries determine the first organic lesion in pulmonary consumption; and when this is taken in connection with the fact that inactivity of the apices predisposes the blood-vessels of these parts to early congestion and rupture, it becomes quite clear why the beginning of this disease is almost universally confined to the apex and not to any other portion of the lung.

Œdema.—Still further evidence that disordered innervation is a prominent feature in pulmonary con-

sumption is furnished us by the pathological condition known as dropsy. This complication generally develops itself most fully during the later stage of the disease, and it is, therefore, regarded as the sequence of cardiac exhaustion or of constitutional collapse. There are good reasons, however, for believing that this is in great part an error, and that the œdema is the only final culmination of a process which had been less obviously active throughout the whole course of the disease. It is quite certain that every practitioner sees cases of consumption in which dropsy supervenes, when no greater circulatory, respiratory, or secretory defect can be detected than had existed for months before the attack manifested itself.

A study of the pathology of dropsy* teaches us that this condition depends on an accumulation of serous fluid in the lymph-spaces of the body. In health these spaces are fed with lymph derived from the capillary arteries, which is absorbed again by the venous radicles and lymphatics. Either the lymphatics or the venous radicles, according to Brunton, are capable of carrying away all the lymph that is poured into the lymph-space by the arterioles, so that in case there is an obstruction in one of the former

*See the interesting article by Dr. Brunton, "On the Pathology of Dropsy," in vol. xxxi, p. 177, of the London *Practitioner*, which mainly furnishes the inspiration for my ramarks on this subject.

channels, the other takes on a compensatory action and performs the work of both. Thus Ranvier, Cohnheim, and Brunton have shown that ligature of the inferior vena cava sometimes produces no œdema whatever in the lower limbs of the dog or the cat. Cohnheim believed the absence of œdema under these conditions to be chiefly due to the establishment of a collateral venous circulation, while Brunton holds that it is on account of increased action of the lymphatic circulation. Both Ludwig and Brunton found that ligature of a vein at once increases the lymphatic stream in that district. As has already been stated, tying the vena cava ascendens does not always produce œdema, but the case is usually different when the sciatic nerve of one side is divided at the same time. Œdema generally follows in this, but not in the opposite leg, although the venous circulation is equally obstructed in both legs. These experiments show, then, that œdema is greatly dependent on want of nerve-power; and that *this* defect is owing to paralysis of the vaso-motor and *not* of the motor nerves, Ranvier, again, showed by cutting the roots of the motor nerves of one limb in the spinal canal before they had joined the sympathetic branches; while in the other limb the sciatic nerve—including the motor and vaso-motor branches—was divided. The limb in which the motor roots were divided was paralyzed, but did not swell, while the other limb became very œdematous. From the results of these

observations we must conclude that the vaso-motor nervous system plays a most important part in the production of œdema, although it must not be overlooked that the state of the blood and the blood-vessels, the impaired sucking power of inspiration, and of the cardiac diastole, also play a subsidiary rôle in bringing about the same end.

Thoracic Pain.—Pain and tenderness in the chest-wall are some of the most common attendants of pulmonary consumption Such patients frequently say that they have always felt weaker on the affected side, and that these are the only symptoms which give them any intimation that there is something wrong within their chests. That the pain is situated no deeper than the parietes of the chest-wall, is evident from the fact that the lungs are not supplied with sensory nerves; nor is it probable that the pain is a mere capricious or incidental accompaniment, without any direct relationship to the disease in the lungs, because it is generally located over the subjacent diseased lung. More than this, it is often found that the whole half of the head or neck, chest, and abdomen, on the side of the affected lung, is more or less hyperæsthetic, or comprises painful spots; while the other half of the trunk is comparatively normal. Such patients, especially of the female sex, are apt to show tender spots along the spine, and may be suffering from what is commonly called an "irritable spine."

By recognizing this phase of the neurotic element in pulmonary consumption, we can also account for the source of the thoracic reflex which is met so frequently, although perhaps not exclusively in this disease. This reflex, which is capable of being excited by immediate percussion of the chest-wall muscles, especially of the affected side, undoubtedly depends on such a peripheral irritability or hyperæsthesia of the intercostal nerves as has been outlined here.

CHAPTER XII.

VAGUS DISEASE THE FUNDAMENTAL LESION IN CONSUMPTION.

From the mass of evidence which has been collected in the previous pages I think it will be admitted by every one that serious disease of the vagi and of the peripheral nervous system is probably always associated with pulmonary consumption. The question arises, however, whether the nerve lesion is primary or secondary, or, in other words, whether the nerve lesion is the cause of the lung disease? Another question, which is virtually involved in the last, is, whether pulmonary consumption—or tuberculosis, as it is preferably called by some—is a separate and distinct pathological entity from its very beginning, or whether it is slowly evolved out of pre-existent pulmonary disease? These two distinct questions are closely interwoven, yet it is hardly practicable to discuss them together at the same time; hence, in order to be able to proceed with the argument, I shall take for granted at present that no hard and fast line can be drawn between pulmonary tuberculosis and other lung affections, and offer the proof for this hereafter.

What evidence have we then for believing that pulmonary consumption depends on a lesion of the pneumogastric nerves? First and foremost we have

the experimental evidence. It has been abundantly demonstrated that section of, or injury to the vagi of animals is followed by a broncho-pneumonia which can barely be distinguished from catarrhal pneumonia in man. Michaelson (*Beiträge zur Untersuchung der Einflusses Beiderseitiger Vaguslähmung auf die Lungen*, von Wittich's Königsberger Physiologische Mettheilungen, 1878, S. 85), concludes from his own researches that bilateral vagotomy is followed by degeneration in the upper lobes, consisting of œdema, hyperæmia, often of splenization, hæmorrhages, and emphysema—all the elements of catarrhal pneumonia, which frequently becomes cheesy. Prof. Schiff also succeeded in developing both hepatization and tuberculization in the lungs of rabbits by the same method as has been stated on a previous page. In a series of experiments of this kind, which I performed last winter, I found yellow tuberculization in one instance in a rabbit's lungs.

The pathological condition which is thus produced has been denominated "schluck" pneumonia in the belief that the pneumonia is brought about by the entrance of mucous, and of particles of food, into the trachea and bronchi of animals in consequence of the paralysis of the pharynx and larynx which is set up by the operation. This does not seem to be true, however, for Arnsperger (*Virchow's Archiv.*, vol. ix, pp. 197 and 437) has shown that if, after section of the nerves, a tube is inserted in the trachea, and the

admission of foreign bodies into the lungs is prevented, the same pulmonary changes occur as before.

At the present time, when almost every disease is traced to bacillary infection, it is interesting to know that pneumonia caused by vagus section contains micro-organisms, which, on being inoculated, reproduce the disease in healthy animals. Thus Jens Schou (*Fortschritte der Medicin*, 1885, No. 15), found that in vagus-pneumonia an abundance of various bacteria were produced in the alveolar and pleural exudations. He isolated three varieties of bacteria which he cultivated in proper media. The first was an elliptic coccus of medium size, which, on being injected directly through the thoracic walls into the lungs, or introduced through the trachea, always generated a typical vagus-pneumonia. Culture fluids, which were sterilized before injection, always produced negative results. The other two were also cocci, differing somewhat in shape from the first, but they were incapable of calling forth the same morbid process in the lungs. Friedländer's coccus could not be found. All the vagotomies were performed in a bloodless manner, under strict antiseptic precautions, and the animals were kept in clean cages and received no food.

These experiments apparently show that ordinary pneumonia is a germ disease, and reasoning from similar premises, one is led into the erroneous belief that pulmonary tuberculosis is of the same nature.

What they really do show is that section of the vagi is capable of developing certain micro-organisms in the respiratory organs which on being injected into the lungs of healthy animals reproduce a disease similar to that from which they sprang originally. Once being produced by vagotomy, the micro-örganisms, if properly infused into the body of healthy animals, have the power of calling forth definite morbid processes in the latter. This principle applies with equal force to the genesis of pulmonary consumption or tuberculosis, as will be learned hereafter, and probably to that of the great majority of other diseases. But in the current discussion of the question of phthisis it is ignored, and this naturally leads to much confusion. If vagus-pneumonia, as has already been shown, and pulmonary tuberculosis, are the natural products of vagus disintegration, and if the micro-organisms, allowing that they are capable of acting as a cause, are merely the results of such disintegration in the first place, it becomes a matter of paramount importance, so far as therapeutics is concerned, if for nothing else, to establish the fact that the disease is not due to vagus lesion before we ascribe it to the agency of a microscopic germ in any given case.

It is quite positively established, then, that catarrhal pneumonia, and even phthisis, may be produced by section of the vagi in animals; and on referring to the foregoing collection of tabulated cases, it will also

be found that the same phenomenon is frequently produced in man in a perfectly natural way through the agency of vagus disintegration. This collection gives a review of the histories of one hundred and fifty-two cases of various stages of pulmonary disease, distributed as follows: Phthisis, 84; pneumonia, 43; bronchitis, 8; œdema, 5; hæmorrhagic infarcts, 4; paralysis of respiration, 4; nature not stated, 4. In these cases the vagi were diseased in 129; in 13 the medulla oblongata was diseased, and in 10 the vag, were not examined. Additionally, Dr. A. Lewin (*Beiträge zür Pathologie der N. Vagus.* Dissertation. St. Petersburg.) investigated the condition of the vagus in twenty cases of pulmonary consumption, and found it diseased in every instance; and Drs. Bianchi and Armandi (*Neurologisches Centralblatt*, 1884, p. 452) found a similarly diseased state of the vagi in eleven subjects who died of paralytic pneumonia. Hence, we now have a definite record of 104 cases of phthisis; 54 cases of pneumonia; and 15 cases of other forms of lung disease, or 170 cases in all, in which the vagi or the medulla oblongata were disorganized.

Now, when we reflect that no one, so far as my researches have extended, except Drs. Lewin, Bianchi and Armandi, has investigated this question with a special view of determining the relationship between diseases of the vagus and that of the lungs, and that all my cases were culled from current literature,

where they had obtained incidental lodgment only, so to speak, it is quite apparent that the two conditions are more intimately connected than is generally supposed, and could be found oftener if search were made in the proper direction.

The dependence of pulmonary consumption on vagus disease, with all its implications, was fully recognized as long ago as 1859 by Dr. Gull, who expressed himself in the following forcible language while discussing two of his cases, to which reference is made in this work. (*Trans. Lond. Path. Soc.*, 3d series, vol. 5, 1859, p. 312.) He said: "This case affords an excellent illustration of the effects which are referable to paralysis of the pulmonary plexus on one side; accumulation of muco-purulent secretion in the paralyzed bronchi, subsequent dilatation of the tubes at their peripheral distribution, concomitant exudation into the air cells, hepatization, and at length disintegration of tissue on the other. * * * It is one of the evils of a too exclusively humoral pathology (*bacillary pathology we might say at the present day**) that leads us to overlook the minute anatomical relations of disease, which are in themselves often a key to the sequence of morbid changes. These cases illustrate this proposition, for the possible local effects on the lung of injury to the pneumogastric and pulmonary plexus being recognized, when-

* That in parenthesis is mine.—AUTHOR.

ever cause for that injury exists we may anticipate its results, and are not wholly dependent upon physical examination as we are if we limit our pathological view to the mere changes in the lung without considering how they are produced."

In his comments on one of his cases, to which I also refer, Dr. Wilks expresses himself in a similar strain. He says (*Trans. Lond. Path. Soc.*, vol. x, p. 1): "It has been said that the patient died of phthisis, and the tumors were found accidentally, but in all probability the affection of the nerves—that is, of the pneumogastric, was the cause of the pulmonary disease, and therefore, so far from neuroma being a harmless affection, it was the cause of the girl's death. This idea was suggested by the observation of several other cases of lung disease occurring in connection with disease of these nerves, particularly as witnessed in aneurism of the aorta and cancer of the œsophagus. In these diseases death is often brought about by the pulmonary affection, and the pneumogastric nerves are found implicated in the disease or pressed on by the tumor."

The testimony which has been collected thus far lends no encouragement to the belief, then, that pulmonary consumption is a disease which is created by a specific virus, but rather that it is a condition which evolves slowly out of pre-existing pulmonary disorders. For it is manifest that in every one of the tabulated histories of cases given in the previous pages the

nature of the lung disease depended on the acuteness or the chronicity of the vagus lesion. Division, injury, or acute disease of the vagi, always resulted in œdema, hyperæmia, hæmorrhage, or bronchitis, but never in phthisis—the last disease only being produced when the vagi were subject to a slow process of devitalization, such as would take place from long-continued pressure, or protracted disease. From these data, one can legitimately infer that all the morbid phenomena occurring in the lungs are but different steps of the same process which may primarily begin in a simple œdema or catarrh, and terminate in pulmonary phthisis.

The fact that phthisis may be reproduced by inoculation in a few days, does not in the least militate against the position taken here, although it is commonly taken for granted that this is unimpeachable evidence in favor of the communicability of the disease from person to person by means of contagium particles. The origination of a disease by inoculation, however, is a thing entirely different from its natural propagation. This is clearly proved by Jens Schou's experiments (see p. 91). Most diseases, as has already been stated, are inoculable, but few are practically contagious or are naturally propagated in this manner. Cancer requires years to mature, yet it may be transmitted by inoculation in a very short time. No one would say from this that cancer is a disease of a few day's growth, or from anything else

that it is contagious, nor would anyone claim that such an experiment gives us the least intelligence as to the normal genesis of cancer. So, on the other hand, a disease may be originated artificially and be propagated naturally from one generation to another, as is demonstrated by the experiments of Dr. Brown-Séquard, who found that the epilepsy which could be generated in the Guinea-pig by section of the larger nerve-trunks, spinal cord, wounding of the medulla, etc., is inherited by the descendants of the animal.

Further proof that the inoculability of a disease leaves us as much as ever in the realm of mystery concerning its natural evolution, is found in the vegetable kingdom, where a graft is known to have the potency of transmitting all its properties not only to the tree on which it is grafted, but also to its seeds. Darwin gives many interesting instances of this kind in his work on *Animals and Plants under Domestication*, and on account of the great relative importance of the subject I shall quote some of the most striking examples which he relates. On page 437 (vol. ii) he says: "It is certain that when trees with variegated leaves are grafted or budded on a common stock, the latter sometimes produces buds bearing variegated leaves; but this may, perhaps, be looked at as a case of inoculated disease." On page 313, same volume, he remarks that "several North American varieties of the plum and peach are well known to reproduce themselves truly by seed; but Downing asserts that

when a graft is taken from one of these trees and placed upon another stock, this grafted tree is found to lose its singular property of producing the same variety by seed, and becomes like all other worked trees—that is, its seedlings become highly variable." On page 473 (vol. i) he further says: "It is notorious that when the variegated jessamine is budded on the common kind, the stock sometimes produces buds bearing variegated leaves. The same thing occurs with the oleander. Mr. Rivers, on the authority of a trustworthy friend, states that some buds of a golden-variegated ash, which were inserted into common ashes, all died except one, but the ash stocks were affected, and produced, both above and below the points of insertion of the plates of bark bearing the dead buds, shoots which bore variegated leaves. Mr. Brown, of Perth, observed, many years ago, in a Highland glen, an ash tree with yellow leaves; and buds taken from this tree were inserted into common ashes, which in consequence were affected, and produced the *Blotched Breadalbane Ash.* This variety has been propagated, and has preserved its character during the last fifty years. Weeping ashes, also, were budded on the affected stocks, and became similarly variegated. Many authors consider variegation as the result of disease; and, on this view, which however is doubtful, for some variegated plants are perfectly healthy and vigorous, the foregoing may be looked at as the direct result of the inoculation of a disease."

These instances illustrate very fully, then, that grafting bears the same relation to the vegetable world as inoculation of disease germs does to that of the animal; and the individual whose intellectual horizon is limited exclusively to a knowledge that plant life can be wonderfully impressed and modified by the operation of grafting, would naturally conclude that the color of the leaves, the nature of the seeds, and all other vegetable properties wherever found, have originated through such an artificial process. There is as much consistency in this reasoning as there is in that of those who hold that the inoculability of a disease is all-sufficient proof that the latter arises through inoculation or contagion under natural conditions. Bearing in mind, then, the possibility of inoculating or engrafting disease, we must never lose sight of the fact that very few diseases are propagated in such a manner, and that the histories of the great majority of the latter bear an impress that they have always been, and still are born, under more natural auspices.

That pulmonary consumption is a consequence of nerve disintegration, is still further proved by the fact that both alcohol and syphilis, on account of their destructive affinity, for the nervous system in general, and for the pneumogastric nerves in particular, have the power of inducing this disease. My collection shows a record of fifteen *post mortem* cases in which lung mischief was associated with alcoholism

and syphilis—twelve of which were phthisical in consequence of manifest degeneration of the vagi and medulla oblongata, and the rest were cases of pneumonia, in two of which the vagi were not examined.

Perhaps the most conclusive proof that pulmonary consumption is a nervous disease, is found in diabetes mellitus. Diabetes is not, according to the most recent returns, believed to be due to any specific micro-organism, as is the case with most other diseases. In fact, it is acknowledged to be not only a disease of the nervous system, but in most cases a disease of that portion of the nervous system which we have seen is essentially involved in the process of pulmonary phthisis, viz.; the vagi and the medulla oblongata. On *a priori* grounds we may, therefore, reasonably expect an intimate association of these two diseases. That this is practically true, is shown by the one hundred and twenty-one cases of diabetes, to which reference has been made, 54 per cent. of which were phthisical. The reason why not every diabetic becomes a subject of phthisis lies probably in the fact that the source of diabetes, as we have seen, may depend more on a peripheral than on a central lesion of the nervous system in some cases, and in these the danger of phthisis is materially lessened. Moreover, since the evolution of phthisis is a slow process, as has been previously shown, it must not be forgotten in this connection, that in severe and aggravated cases of diabetes the patients

die before phthisis has time to supervene. Dr. Mackenzie very pertinently says (p. 47, *supra*): "It would seem, indeed, that the natural mode of termination of diabetes is by inducing phthisis, but that in a large proportion of cases this stage is only partly reached, or not reached at all, owing to the patient being prematurely cut off by coma."

The evidence which has thus far been culled from the history of diabetes shows very strikingly in favor of the neurotic origin of pulmonary consumption. Topographically, it may be said, then, that the fundamental lesion of both diseases belongs to the same special region of the nervous system. It is occasionally said, however, that diabetic phthisis is not a true tuberculosis, because the characteristic bacillus is not present in the lungs. This assumption may be laid to rest, for the specific micro-organism has been found in this form of phthisis by Immermann, Leyden, Merkel, Riegel, and others (*Schmidt's Jahrbücher*, Band, 220, S. 83).

That which is true of alcohol, syphilis, and diabetes in this respect, also holds true of beri-beri and leprosy. The symptoms, pathology, and morbid anatomy of both of these diseases show that their essential lesion is in the peripheral nerves, and the reason why they are so frequently associated with pulmonary consumption, as has been shown in the table, is because the vagi become involved in the process of nerve degeneration. Both are believed by some to

be propagated through the agency of specific micro-organisms. It is doubtful, however, if it were not the fashion to ascribe almost every disease to the pathogenic action of a bacillus, whether any suspicion of the kind would ever be entertained.

At this stage of the discussion the question may present itself to the reader's mind: Whether, if phthisis accompanies so many varied morbid conditions as it is represented to do here, it may not arise in consequence of *any* general exhaustion of the body? In answering this question, it may be stated that, as a complication, pulmonary consumption is almost exclusively found among diseases which are connected with the nervous system. On the other hand, it is but rarely met in small-pox, or scarlatina, or in any of the contagious diseases, except in those which directly implicate the respiratory system of nerves. The same is also true of cancer, a disease which produces a profound and hopeless depression of the constitution; yet pulmonary consumption is one of its rare allies. Walshe, in his work *On Diseases of the Lungs*, 1871, p. 518, states that "although there is no absolute constitutional antagonism between the two complaints, they rarely coexist." These facts, in connection with others which could be presented, show, then, that phthisis does not follow in the wake of every form of bodily exhaustion, but requires for its development, in the vast majority of cases, a special exhaustion of the nervous system.

CHAPTER XIII.

THE PATHOLOGY OF TUBERCLE.

True tubercle is a genuine growth. Nominally there are two varieties of tubercle—the yellow and the grey. The yellow variety, only being the accumulated product of a catarrhal infiltration, is not a tubercle, and therefore the grey variety, which is a connective-tissue hyperplasia, is the only structure deserving of that name. This is a non-vascular, cellular nodule, containing giant cells, and is associated with tubercle bacilli, and after attaining a certain stage of growth it uhdergoes caseous degeneration. The yellow tubercle is likewise associated with bacilli and also undergoes caseation. In fact caseation is the natural mode of death of any albuminous material which is slowly cut off from its source of nutrition. Neither kind of tubercle contains blood-vessels, hence they receive their pabulum from their immediate environment. If, as in the case of the grey structure, the growth becomes too large, or, as in the case of the yellow variety, the aggregation of catarrhal products infringes too much on the surrounding circulation, both forms soften from the centre toward the periphery.

Such being the nature of tubercle, what is its mode of genesis? It has already been shown from experimental evidence that irritation of the vagi leads

to pneumonia. This condition implies at least a capillary engorgment, and a rapid multiplication and accumulation of epithelial cells in the alveoli. The alveoli thus filled up constitute what is commonly known as yellow tubercle, and when a whole cluster of these structures is involved in this way they form a nodule which in due course of time becomes isolated from the circulation, softens by caseation, and is finally expelled, leaving a cavity behind. Now, it is very important to remember that during caseation a virus develops which has the power of producing genuine tubercles. This poison is absorbed and transported by the lymphatics, and it is in these vessels that the first evidence of tubercle formation appears; and to these structures this process is almost entirely confined—showing, therefore, that, in this case at least, tubercle is an infectious product. When the caseating centre is located in the lungs the infection may be limited to a part or to the whole of these organs, but when this is situated in another part of the body the infection may be general, as is shown by the history of the following case, which, on account of its intense interest, as well as on account of the care and accuracy with which it has been investigated, I take the liberty of quoting from Dr. Hamilton's excellent work on the *Pathology of Bronchitis, Catarrhal Pneumonia, and Tubercle* (p. 166), and to which I am deeply indebted for many valuable ideas on the subject under discussion. He says:

In the following description of primary tubercle in the lung, I shall take as my guide a typical instance of the disease in which its commencement, course and duration were accurately known. The subject of it was a woman aged twenty, who was delivered of a child thirty-three days before death. Previous to this she had, from all accounts, been in good health. On the seventh day after delivery, she suffered from a rigor, followed by considerable fever, which continued from this time up till that of death. It was also evident from the symptoms that the patient was suffering from peritonitis. The only pulmonary symptoms were those of slight bronchitis. She rallied to a certain extent at one period of the disease, but about seven days before death became worse, and finally died with signs of cerebral meningits.

The dates of the case are important as bearing upon the age of the tubercular deposits found in the lungs; her illness dated from a week after delivery, and twenty-five days elapsed from this before death occurred, so that, even at the utmost, a month had been sufficient to induce all the morbid appearances to be described, and the tubercular growths in the lungs could not have been more than from a fortnight to three weeks old.

After death there was found to be extensive peritonitis, of quite recent and also of somewhat older date. In many places, more especially behind the uterus, the peritonitic lymphy effusion had become caseous, and here and there this had undergone softening. Covering the peritoneum, more especially in regions adjacent to the softening caseous effusion, there were large numbers of grey tubercles in the peritoneum, running in lines along the course of the lymphatic vessels leading to the under surface of the

diaphragm. Nearly all the organs showed tubercles of recent origin, and there was extensive cerebral tubercular meningitis. The lungs presented appearances typical of those usually found in cases of primary tubercle, and it is specially to these that I must now direct the reader's attention.

There was no evidence of recent pleurisy. In looking over the post-mortem accounts of many similar cases of primary tubercle of the lung, I find that the records, in regard to this particular, vary. In certain instances there has been a little fibrinous deposit on the pleura, but, usually, it has either been absent, or has been present in comparatively small amount. A little roughening and dulness of the pleural lustre has been the utmost I have observed in any such case.

The pleura was beset with tubercle nodules, and when incised they could be seen to lie in its deep layer. Similar tubercle nodules were seen in immense abundance in both lungs, uniformly distributed throughout all the lobes. They had the same characters as those seen in the pleura. They were round and had a sharpely defined border, which was abruptly marked off from the surrounding pulmonary parenchyma. Their color was grey, and they had a somewhat gelatinous aspect. They were about the size of a mustard seed. This is generally the case both in primary and in secondary tubercle of the lung. Bodies having an aspect similar to that of tubercle, but larger than a mustard seed, usually prove to be groups of air vesicles in a state of catarrhal pneumonia. All the nodules were, as near as possible, of the same size. They either ran in lines along the course of a small branch of the pulmonary artery, or they were aggregated in little clusters. The

former was the commoner of the two arrangements. There was not any avidence of the nodules uniting to form large nodules; for even although they might occasionally be seen in groups, yet the individual members of the group, after their border was once defined, never coalesced so as to constitute a single mass, and they never increased in size beyond the dimensions above stated.

Some of the tubercles occasionally had a slight cream-yellow color, but this was not their usual appearance, a grey connective-tissue-like aspect being that which was most general. There was a total absence of blood-coloration within them, and hence they stood out prominently from the highly vascular background on which they lay.

In this case the commencement of the disease evidently was peritonitis following delivery. Caseation ensued in the peritonitic effusion; thus softened, the caseous debris was absorbed, and gave rise to the formation of tubercles in various organs. The tubercles in the peritoneum were evidently of local formation, and had their origin in the lymphatics. In the other organs, there is every reason to believe from the histological examination, that the blood-vessels were the means of transmission of the caseous products, and that the tubercle was formed within them.

It is evident from the account of this case, and from many others which might be cited having a similar history, that well developed tubercles may originate within a space of from two to three weeks. Tubercle was formerly considered to be a chronic disease, and the fact of giant-cells being developed in so short a time as three weeks has partly led M. Charcot to the erroneous conclusion that catarrhal

pneumonia and tubercle of the human being are alike.

When we find that tubercle growths can be artificially developed within a few weeks in animals as a result of injection of caseous material, there is no difficulty in seeing how they may originate in a similar short space of time within the human subject, where practically speaking, the conditions essential for their production are the same. Secondary * tubercle of the lung no doubt, is occasionally a very chronic disease, but the primary form runs an acute course, the tubercle-nodule being well developed in from a fortnight to three weeks.

We may therefore take it for granted that when a caseous centre exists in the body, or in catarrhal pneumonia, bronchiectasy, pelvic cellulitis, or in a cheesy bronchial or cervical gland, tubercular infection may always be legitimately expected. It must be remembered, however, that tubercle in itself is not so deadly as it seems to be when judged from the standpoint of acute pulmonary tuberculosis. This disease is of comparatively infrequent occurrence, and it would not possess such a great fatality were it not that it involves and overpowers organs, the full integrity of which is absolutely essential to life. This statement is founded on our knowledge of what takes

* Primary tubercle, according to Dr. Hamilton, is that variety of the product which is produced by a caseous infecting centre without; and secondary when this is located within the lung. Essentially Dr, Hamilton's views coincide with those of Virchow and others on this subject.

place in tuberculosis of other parts of the body, notably in leprosy and in tubercular peritonitis. In the former disease the tuberculous process may exist in the skin for from twelve to eighteen years, if the patient is not carried off by some intercurrent malady, like pulmonary phthisis, or something else. And, indeed, there are recorded instances in which the disease disappeared entirely. The same is also true so far as tubercular peritonitis is concerned. Our gynæcological brethren bring us the most unmistakable proof that the disease has been entirely cured by opening and thoroughly cleansing the abdominal cavity.* The harmlessness of tuberculosis is still further brought to mind by the dissecting tubercle which is so often contracted while making post-mortem examination of tubercular bodies, and in most cases without the least detrimental influence.

Tubercle formation is really a conservative process, ultimately tending towards the production of fibrous tissue, and must be regarded more in the light of a constructive than of a destructive process Yet it is true that if tubercles form too rapidly, or too profusely, they undergo caseation and create cavities like those which follow the yellow or catarrhal variety; but this, it must be admitted, is a rare exception.

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*Laparotomy for Tubercular Peritonitis, with Report of One Case. By Dr. George E. Shoemaker, Medical and Surgical Reporter, April 13th, 1889, page 447.

In regard to this subject, Dr. Hamilton (loc. cit. p. 206) says:

Does a primary uncomplicated eruption of miliary tubercle, such as one sees in children resulting from some distant caseous infecting source, lead to excavation of the lung? I have never seen it. When softening occurs, a microscopic cavity may result. Such cavities, however, do not run together to constitute larger ones, as is the case in catarrhal pneumonia. But the softenting takes place locally, and as soon as the centre of the tubercle has been absorbed by the lymphatics, the peripheral part contracts and the space which has been left is obliterated. The tendency of the tubercles in such a lung, if the person lives long enough, is to become fibrous and to give rise to a cirrhosis of the organ. What I wish specially to emphasize is that uncomplicated tubercle of the lung does *not* cause excavation, and hence that the term tubercular phthisis, when used in this sense, is erroneous and misleading.

From what has been stated, it is quite obvious that of the two varieties of pulmonary consumption—the catarrhal and the tubercular—the morbidity and the mortality of the former far exceed those of the latter. And now, before concluding this chapter, it will be of interest, in this connection, to institute a comparison between the course of pulmonary consumption and that of leprosy, with a view of showing their analogous relations. Moreover, since the pathology of leprosy is pretty thoroughly established, such a study will also serve to give us a better understanding of the origin and nature of pulmonary consumption.

Although leprosy is divided into two varieties,—the anæsthetic and the tubercular—and is, therefore, like pulmonary consumption, a duplex disease so far as its outward manifestations are concerned, there are, as has already been shown, good reasons for regarding both varieties as being primarily a disease of the peripheral nerves. This is universally admitted in the case of the anæsthetic variety, and it is also freely admitted that the tubercular variety is only another expression of the same disease; hence, it must be true that that which causes one must cause the other. This opinion is further substantiated by the fact that the two varieties are frequently combined in the same individual.

The pathology of leprosy, as obtained from the works of Hillis and Bidenkap, has already been briefly described (see page 55), and only those points will be reiterated here which have a special bearing on this part of the discussion. The anæsthetic variety is characterized by a cutaneous eruption, consisting of moleculæ, vesicles, bullæ, and finally ulcers. Some time before the eruplion appears there is evidence of nerve disease. The skin covering the affected part becomes numb along the course of certain nerves, which swell and can be felt through the skin. The superficial ramifications are first implicated, after which the larger trunks become involved. As a rule the nerve centres escape. Here then exists a morbid process which to all intents and purposes is a catar-

rhal inflammation of the skin called forth by disorder of the peripheral nerves. Now, a catarrhal inflammation of the skin finds its analogue in a catarrhal inflammation of the mucous membranes; and it is perfectly reasonable to believe if one is due to perverted nerve influence, that the other may be due to the same cause. This relation, so far as the lungs are concerned, is strikingly corroborated by the experimental and clinical evidence already set forth in which injury and disease of the vagi gave rise to catarrhal pneumonia, bronchitis, and phthisis.

In the tubercular form of leprosy, genuine eruptions of tubercles occur in the skin, which contain giant cells precisely identical with those found in pulmonary tuberculosis. These tubercles are also associated with a bacillus which resembles the tubercle bacillus so closely that the two can only be differentiated with difficulty, and, indeed, by some they are believed to be identical. In reference to a distinction between these micro-organisms, Dr. Robert Koch says that while the tubercle bacillus bears the most striking resemblance to the bacillus of leprosy, so far as form and size are concerned, the latter is a little more pointed at the ends than the former.* On this point Dr. Baumgarten, an equally eminent authority, says that the color-reaction of the two is the same,

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* Die Aetiologie der Tuberculose. Berliner Klinische Wochenschrift, 1882, S, 221.

the *only* difference being the length of time necessary to discolor each micro-organism.† But Gottstein states that the resistance to the discoloring process is equal in the two kinds of bacilli.‡ Whatever the truth may be, it is quite clear that the difference between these two structures is infinitely small, and raises a strong presumption that tuberculosis and leprosy are more closely allied in nature than is currently supposed.

Since both varieties rest primarily on a similar pathological condition of the peripheral nerves, it is strange that one should give rise to an anæsthetic, and the other to a catarrhal manifestation. That there is not an impassable gulf between these two states, however, and that they merely represent different sides of the same lesion, is shown by the facts that if the patient lives long enough, one form always runs into the other, and that they are frequently intermixed in the same patient (Danielssen, Boeck, Carter, and Shaw). One thing seems very certain, and that is, the two forms develop with different degrees of intensity, and consequently move with different degrees of rapidity. The tubercular is always more active and of shorter duration than the anæsthetic

† Uber die Untersuchungsmerkmale der Bacillen der Tuberkulose und der Lepra. Monatshft. f. Prakt. Dermatologie, Bd. 3, S. 193.

‡ Bemerkungen über das Farbungwerhalten der Tuberkel-bacillen. Deutsche Med. Wochenschrift, 1886, No. 42.

form—thus again showing a parallel to the two forms of pulmonary consumption. The reason why the former is more fatal than the latter is probably not so much on account of the tubercular lesion of the skin, as on account of the secondary invasion of the lungs, intestines and of other important organs. Miliary tuberculosis of the lung is always acute and short lived, as has already been intimated, and is regarded as a disease the existence of which is dependent on a neighboring caseating centre. But in tubercular leprosy the tubercular eruption is one of the first outward manifestations of the disease and occurs without any obvious caseous contamination.

Now, in the face of the probability that this may take place independently of any caseous infection, and without in the least doubting the infectiousness of the virus of tubercle, it is legitimate to inquire whether or not tubercle may, under some conditions, be elaborated through the direct intervention of the nervous system? This is especially pertinent when we reflect that tubercle is a fibrous product, and that the researches of v. Recklinghausen, Esmarch, Kulenkampff and Kriege * seem to point out that the peripheral cutaneous nerves play an important part in the production of multiple fibromata and other fibrous growths. The adoption of the bacillus theory will

* Dr. H. Kriege. Ueber das Uerhalten der Nervenfasern in den Multiplen Fibromen der Haut u. in den Neuromen. Virchow's Archiv. Band 108, S. 466.

not in the least aid us in solving this difficulty. For if we accept the bacillus lepra as the cause of the nerve disease underlying the tubercular eruption, we are still confronted by the question as to why the anæsthetic form, in which the bacillus also prevails, is not characterized by tuberculization? So far as I am able to perceive, a specific germ can only originate a specific product, as when thistles are sown it is useless to expect a crop of corn. But if, as has been suggested, the peripheral nerves play a causative rôle in the cutaneous tuberculization of leprosy, then the two-fold manifestation of this disease may possibly be explained by the difference in the intensity and activity which exists between the two morbid processes.

CHAPTER XIV.

THE BACILLUS AS A CAUSE OF CONSUMPTION.

Pulmonary consumption may arise from causes which reside either within or without the body. The bacillus theory maintains that it emanates from without, and is propagated from man to man through the medium of a specific micro-organism called the bacillus tuberculosis, and hence is a contagious disease. Contagiousness involves the acceptance of the correlative idea that, if other things are the same, all those exposed to the contagion become equally liable to the disease. The susceptibility to contagious diseases may, therefore, be very aptly compared to the happenings of railway accidents. While, owing to unlike, conditions, only a portion of those exposed to such calamities are injured or killed, it is nevertheless true that the mortality rate from railroad accidents is higher among those who are than among those who are not exposed to them. In the application of this it must be borne in mind that the causes of disease which operate from the inside are far more complex, and vastly more difficult of comprehension than those which operate from the outside, and therefore in diseases due to the former the single cause of contagion is displaced by the important influences of age, sex, food, inheritance, diathesis, temperament, occupation, climate, etc. These principles are obviously so funda-

mental to the free and fair discussion of this question that I trust it is not necessary to say anything further in their defence; and we will now proceed to examine them in the light of all the knowledge which can be gathered on this interesting subject.

From what has already been said in the foregoing pages, it is quite clear that pulmonary consumption may be propagated from man to man, or from man to animals, by means of inoculation. This is probably true of all diseases. But is it a scientific deduction to hold, that because a disease is communicable in this way, it must also be maintained in the same way in the human family under ordinary practical conditions? I feel convinced that this is a most illogical position to assume in this matter. It would be just as true to claim, that because a disease cannot be thus reproduced, it is not a real contagious disease. This is clearly the case with whooping-cough — an undeniable contagious disease. Micro-organisms of a rod-like form, which can be isolated, cultivated, and tinged with methyl-violet, have been found exclusively in the sputum of those suffering from whooping-cough, yet it is doubtful whether the genuine form of this disease has ever been transmitted to animals through the artificial introduction of these micro-organisms. (See *Zur Frage der Keuchhusten-Bakterie*, von Dr. G. Ssemtschenko, Petersb.: Med. Wochnschr., XIII, 22, 23, 1888; Schmidt's Jahrbucher, Bd. 220, S. 12; C. Burger, *Der Keuchhustenpilz*, Ber-

liner Klin. Wochenschrift, 1883, No. 2). Laboratory experiments, therefore, show that whooping-cough is not a contagious disease, yet there is ample ground for believing that it will always be regarded as such, no matter how much negative experimental evidence accumulates against it in this respect. Hence it is very clear that assumptions, either for or against the contagiousness of any disease, which rest on mere theoretic foundations, are not only unfortunate in that they clash with firmly-rooted clinical experience, but because they are calculated to bring scientific laboratory work into disrepute. The truth is, that practical medicine, and not the laboratory, is the avenue through which this matter must be fully and finally determined.

From our original premises it is certain, then, that if pulmonary consumption is propagated through contagion, it must prevail to a much larger extent among those exposed to its virus than among those not so exposed. Investigations show that the bacilli, which are presumed to be the contagium particles, abound in those localities where the disease exists, and are absent where the disease is not found. Such localities are hospitals for consumptives, and the homes of those who suffer from the disease. It is vain to make the specious plea that in modern hospitals for consumption the dangers of infection are greatly reduced by cleanliness and ventilation, for the fact remains, that in spite of these precautions,

the bacilli abound in these places. It is quite natural, therefore, to expect, under these circumstances, that physicians, nurses, and attendants of consumption hospitals, and relatives of consumptive patients, are specially liable to the disease. Let us examine this question from a practical standpoint.

In connection with this part of the subject, it is of interest to note that efforts have recently been made by Dr. Cornet, of Berlin, to show that the consumption rate is very high among the inmates of convents, a considerable number of whom are engaged in nursing consumptives and sufferers from other diseases, while others are employed in teaching. These investigations, which attracted considerable attention at the time of their publication, because they were regarded by some as being a crucial experiment, really only show what has been known long ago, viz., that the consumption rate is greater among the inmates of closed institutions, like convents, prisons, etc., than it is among people who lead an out-door life. His researches would have been of some value had they comprised a table of the comparative liability to phthisis between those who nurse consumptives and those who nurse patients affected with other diseases, as well as a table comparing the liability between the former class of nurses and those inmates who are engaged in teaching. This would have reached the pith of the point at issue, and it is a source of great regret that Dr. Cornet has entirely ignored it in his work.

How do those fare, then, who are almost constantly exposed to the influence of tubercle bacilli? In the first place, is there any overwhelming instinctive feeling, apart from mere theory, which prompts such persons to regard pulmonary consumption as a contagious disease? On this point, and among other things, Professor Lichtheim expressed himself, before the Second Medical Congress of German Physicians, in 1883, in substance as follows: There are many facts which contradict the contagious nature of pulmonary phthisis. Intercourse with consumptive people is not attended with danger. We hospital physicians spend much time among such people, and we have such confidence in the innocuousness of the disease that we do not hesitate to mix healthy persons with them in institutions devoted to its treatment, and we must confess that this procedure is practically unattended by any unfavorable results. He further stated that the mortality of the attendants in the principal hospitals for consumption was surprisingly low; and that he could, from investigation, endorse the belief that there is no relation between the increase of the number of phthisical patients at a health resort and the number of deaths from consumption occurring among the native inhabitants of the locality.

From the following, which is copied from Dr. Schnyder's valuable paper (see infra, p. 131), and which shows the average annual number of deaths

from consumption for four years, in various occupations, proportioned to each 1,000 living, it will be seen that among physicians, who naturally come in almost daily contact with the disease, the phthisis mortality is lower than that of any other occupation:

	AGES.			
	20–29	30–39	40–49	50–59
Physicians	4.81	4.67	5.28	3.22
Lawyers and jurists	4.92	4.77	4.32	6.47
Butchers	5.59	6.82	5.85	6.29
Coopers	3.32	8.68	7.09	4.55
Locksmiths	5.35	7.29	10.37	11.60
Stone-grinders	3.01	8.65	9.90	12.60

The statistics of the Brompton Hospital for Consumption, in London, as collected by Drs. Williams and Humphreys, speak in the same emphatic manner against the contagiousness of pulmonary consumption. This large institution shows that during a period of thirty-six years not a single clearly authenticated case of pulmonary consumption among all its attachés emanated from within its walls. During the thirty-six years there were twenty-nine physicians and assistant physicians connected with the hospital, and among these occurred a single case of phthisis, and he was tuberculous before he entered it. The rest were all well. Dr. Edwards was resident for twenty-six years, but showed no sign of the disease.

During that period there were employed one hundred and fifty clinical assistants. Of these, eight died of consumption; but all, except one, were sufferers from the disease before they became connected with the hospital, and in this case there is some doubt as to the time of the origin of the trouble. Among the one hundred and one nurses, of which there is a health record, one was suffering from a slow form of the disease, which may have been contracted during her stay at the hospital, although she was predisposed to consumption. She was an old employé, and able to attend to her duties efficiently most of the time. No more positive proof of the non-contagiousness of pulmonary consumption could be gathered anywhere than is furnished by this large charity. Everyone of these individuals associated with this hospital, many of whom are still alive, were almost constantly exposed to the disease, and still there is only a single case to which the least claim of contagion can be made, and this rests largely on suspicion.

The products of these statistics are strikingly confirmed by those which relate to the influence of the Consumption Hospital of Görbersdorf in Germany on the death rate from phthisis among the inhabitants of that town. Dr. Brehmer, who has been in charge of that institution for twenty years, says *

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* Die Aetiologie der Chronischen Lungenschwindsucht, p. 18.

that since the year 1854 more than ten thousand consumptives resided in the Görbersdorf Hospital, who daily walked the streets of the town and commingled with its inhabitants. The latter were, therefore, continuously respiring an atmosphere more or less laden with tubercle-bacilli emanating from the dried expectoration of these consumptive visitors; yet, in spite of these favorable conditions for contagion, the mortality statistics of the town show that prior to 1854 there were 10.07 annual deaths from consumption among its inhabitants, while from 1854 to 1880, there were only 5.0 deaths per year, or a death-rate somewhat less than one-half of what it was before the introduction of the hospital. Dr. Brehmer, and justly I think, ascribes this diminished consumptive rate to the improved facilities for money getting, and to the lessened bodily exposure implied by this, which the inhabitants of Görbersdorf enjoyed since the establishment of this institution. These figures are especially interesting in view of the assertions frequently made that the healthful influence of mountain resorts is impaired by the infectiousness of the exhalations and expectorations coming from consumptive people, who go there for the purpose of seeking relief, showing that such assumptions are more fanciful than real.

Furthermore we have the statistics of Friedrichshain Hospital in Berlin, recently gathered by Dr. Fürbinger, which show that during a period of six-

teen years out of 459 male nurses there were 4 (2 of whom were tuberculous before entering); of 339 female nurses there were 2; of 83 physicians there were 3 (one of whom entered with the disease); who became consumptive. Of 108 Victoria sisters, who were engaged as nurses in the institution from two to five and a half years, only one became tuberculous.

Such exemption does not obtain, as is so often asserted, in hospitals devoted to the treatment of infectious diseases. This is well shown, at least so far as typhoid fever is concerned, in the records of the Massachusetts General and the Boston City Hospitals. In the former—from 1882 to 1887—no less than seven, and probably eleven; and in the latter—from 1884 to 1888 – twenty-eight cases of typhoid fever occurred among the medical attendants and employés of these institutions.†

Contagion Between Husband and Wife.—When inquiry is made concerning the contagiousness of pulmonary consumption between husband and wife, or between members of the same family, etc., a lack of evidence favorable to the contagion theory is found. In 1883, a committee, appointed by the British Medical Association, investigated the contagiousness of this disease by sending out printed circulars asking the members of the profession through-

† Boston Medical and Surgical Journal, May 24th, 1888, pp. 513 and 523.

out England whether they had observed any cases in which the disease was believed to have been communicated. The committee received ten hundred and seventy-eight answers. Of these, seven hundred and seventy-eight were negative, thirty-nine were doubtful, and two hundred and sixty-one were affirmative. Of the affirmative answers, one hundred and fifty-eight related to cases where the communication of the disease was supposed to have taken place between husband and wife, eighty-one between members of the same family, and the remainder were principally cases between which there was no blood connection.

On account of the large number of affirmative answers, this report has been made to subserve the interests of those who believe in the contagiousness of phthisis. Evidently this is unfair, since the aim of the investigation was not to ascertain the number of absolutely well demonstrated cases in which contagion was present or absent, for this would obviously have been next to an impossibility, but it was to collect the individual opinions of a large number of physicians as to whether they believed the disease to have been contagious in certain cases or not, and this resulted in seven hundred and seventy-eight negative, and two hundred and sixty-one affirmative votes. Are we, therefore, justified in assuming that the two hundred and sixty-one opinions are of more weight than the seven hundred and seventy-eight negative

ones, and thereby imply that the former only had the fortune or the misfortune of meeting cases which originated through contagion, and the latter had not? Is it not more probable that all of them witnessed cases around which hung a cloud of suspicion that they might or might not be contagious, but that seven hundred and seventy-eight did not consider the proof strong enough to outweigh that which in their minds was in favor of other and more powerful influences in the causation of the disease?

As still further proof of the correctness of the view concerning the non-contagiousness of phthisis between husband and wife, I beg to call attention to a most admirable paper, entitled *Eine Statistische Studie als Beitrage zur Aetiologie der Lungenschwindsucht*, contributed by Dr. Schnyder to Nos. 10, 11 and 12 of the *Correspondenzblatt für Schweizer Aertzte* for 1886. The substance of this paper is based on three thousand four hundred and sixty-one cases of pulmonary phthisis, which were observed by its author while resident physician at the health resort of Weissenburg. Of these cases, eight hundred and forty-four occurred among married people, but in four hundred and forty-five of them it was the husband only, and in three hundred and sixty-seven instances it was the wife only, while in thirty-two cases both husband and wife were affected. Now, it must be admitted by the most ardent contagionist that here existed very favorable conditions for a propagation of the disease

between husband and wife, through contact; and the fact that eight hundred and twelve of these cases escaped a suspicion of contagion, shows clearly that the disease is not by any means readily communicated even under the most extraordinary circumstances. But is it beyond doubt that the thirty-two cases, in which both husband and wife were affected, originated through inter-communication? I think not, for, as Dr. Schnyder says, it is a notorious fact that, in spite of all entreaties and warnings, young people are often wedded while suffering from active lung disease. He relates four such cases from his own experience in which both the bride and bridegroom came to Weissenburg, fresh from the matrimonial altar, to be treated for phthisis, from which they both suffered. Aside from those who are wedded while suffering from active phthisis, there is no question that many young people of both sexes carry a hereditary taint of the disease in their constitution, which only awakens from its slumbering condition to assert its power when the many varied burdens and demands of family life begin to exhaust the vital resources.

On the communicability of phthisis between husband and wife, the late Dr. Flint, in his work *On Phthisis* (p. 420), says: "In my collection of cases (670) these five are all that I find in which there is room for the suspicion of the disease having been communicated from the husband to the wife, or the

wife to the husband. By making inquiries of members of the medical profession, and searching periodicals, doubtless a considerable number of similar cases might be obtained. Collected in this way, however, they would not prove communicability. According to the law of chances, a disease of such frequent occurrence as phthisis would affect in succession a husband and wife, or *vice-versa*, in a certain proportion of cases. Conceding that the histories of some of my cases are defective in information on this point, it is certain that the instances in which transmissibility may be suspected are not sufficient in number to be not allowed for as coincidences. It must, therefore, be concluded that the analysis of my cases does not furnish facts sufficient to render the communicability of phthisis probable."

The studies of M. Leudet (Medical and Surgical Reporter, Feb. 1, 1890, page 142,) gave similar negative results. He investigated the fate of the surviving person, in case of death by phthisis of either the husband or wife, for a period of twenty-five years following the death of one of the married persons. This shows that out of 112 widows and widowers of certainly phthisical patients, by far the majority of them escaped entirely. Out of the 112 survivors only seven (four women and three men) became phthisical, hence there remained 105 who lived intimately with tuberculous persons without contracting the disease.

Evidence of infection between married people is,

therefore, less plentiful than it is supposed to be in some quarters. But were the fact of the communication established in any given case, it would still devolve on the contagionists to show that the virus was not introduced under very unusual conditions—such as being inoculated through an abrasion of the skin or mucous membrane by intimate contact of one person with another.

House Infection.—The possibility of contracting phthisis by inhabiting houses in which consumptives lived and died, has recently been investigated by Dr. Lawrence F. Flick, of Philadelphia, who embodied the results of his researches in an elaborate paper which he read before the meeting of the Pennsylvania State Medical Society in 1888. His investigations were confined to the Fifth ward in this city, and extended over a period of twenty-five years, and they show that nearly all the deaths from consumption within that area and time were confined to certain groups of houses, and from these premises he feels himself justified in concluding that houses in which consumptives die are dangerous to their occupants, and that phthisis spreads by contact.

It is quite obvious that the enormous labor which has been expended on Dr. Flick's observations is out of all proportion to the benefit which it will yield to scientific medicine. What he does demonstrate is, that the deaths from phthisis take place in certain localities in a given area, but he does not show

whether this is a mere coincidence, or whether it is brought about through the influence of contagion. How does he know that injuries, insanity, and other diseases, which are regarded by all as being not contagious, may not also be grouped in the same manner? In the discussion which followed the reading of the paper, Dr. F. P. Henry plucked out the gist of the argument of this paper when he said* that, according to Dr. Flick's remarks, "I infer that the poison of phthisis may linger in a house for a long period, and give rise to the disease in those successively inhabiting such a house. I think that precisely similar facts might be proved with reference to syphilis. For instance: Sailors suffer very largely from syphilis; sailors are usually in ships at sea; it might therefore be argued that the poison of syphilis exists in the sea air and lingers in the fabric of ships. Such a line of argument could easily be reduced to absurdity. The facts brought forward in this paper seem to be capable of explanation as coincidences. In a number of the houses in the ward referred to, there have been four or more cases at different times, but it does not necessarily follow that the poison was lurking in these houses. The study of each case might show that it had its origin apart from the residence of the patient."

Much more direct and conclusive evidence of

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*See Trans. of the Med. Soc. of Pennsylvania, 1888, p. 186.

this character was collected by Professor Langerhans* concerning the liability of the inhabitants of the island of Madeira to pulmonary consumption. For two hundred years this island has been under the social domination of the resident English colony, of which there exists an accurate statistical health record since the year 1536—at which time the colony contained two hundred and ten inhabitants. As is well known, this island has been a famous resort for consumptives for the last hundred years, and is now annually visited by from three to four hundred of this class of sufferers. These people, on account of a peculiar custom, are not accommodated in hotels, or in institutions specially adapted for their use, but they either live in rented houses, or are lodged and boarded by resident families. Hence there is not a house or a family in this whole colony which has not been at one time or another subjected to the continued influence of consumptive people. Professor Langerhans, who was himself a sufferer from the disease, was a practicing physician on this island for nine years, and was consequently well acquainted with his surroundings and he applied himself to the task of determining the destiny of those residents who were brought into such intimate social contact with sufferers from consumption.

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* *Zur Aetiologie der Phthisie.* Virchow's Archiv., 1884, Band 97, S. 289.

Of the two hundred and ten English colonists who lived here in 1836, there were alive eighty-six, and dead one hundred and twenty-four, in 1883. Of those dead, four died from phthisis—in two of which cases the disease was contracted here, while in the other two it came on while the individuals were absent from the island. In another table he shows that from 1836 to 1864, eighty-three healthy individuals became members of the colony. Of these, thirty-three died, but not a single one from phthisis. More than this, in a third table he shows that from 1836 to 1864 there were born in the colony, from healthy parents, one hundred and forty-seven children, of which twenty-eight died—one of which from phthisis. Of the one hundred and nineteen living, none are phthisical. The consumptive died at the age of 37, and was strongly addicted to alcohol.

Now when we realize that only five, and probably only three, deaths occurred from consumption out of a population of four hundred and forty, and this after being constantly exposed for many years to hundreds of those suffering from this disease, it should be pretty thoroughly established that the intermixing of consumptives with healthy people in the same houses even, is not fraught with such awful consequences as one would be led to believe from the arguments of our friends on the other side of this question.

Seed and Soil Theory.—The fundamental pre-

mise laid down at the outset of this chaper, viz.: that all are equally liable to a contagious disease if the conditions are the same, carries with it an implication of some sort which must now receive attention in order to round off this part of the discussion. The external conditions which have just been examined tell very decidedly against the propagation of pulmonary consumption by means of a virus introduced from the exterior, but those who adhere to the contagious theory claim that the bacillus is powerless to attack the body unless a suitable soil is present in the tissues on which it is supposed to operate, or in other words, there must be a certain degree of disease, or at least a predisposition present before the bacillus can be received into the body and exert a pernicious influence on it. Apart from the fact that this admission virtually destroys a belief in the potency of the bacillus as an all-sufficient cause of pulmonary consumption, it would, if it were true, demonstrate that the bacillus tuberculosis stands isolated in this respect. Do syphilis, small-pox, etc., require a specially prepared soil before they become active disease producing factors, or do they not, with a great degree of certainty, attack the unprotected on their first exposure? Now the whole fabric of the contagious theory of phthisis rests on the knowledge of the transmission of tuberculosis from man to the lower animals through inoculation, and what warrant can be drawn from this for the opinion that the tubercle

bacillus requires a special nidus before it is capable of developing phthisis? If this were true, it would also be necessary for the experimenter to calculate on a proper soil in his animals before operating, but instead of this he possesses the most perfect confidence in his ability to call forth at any time, and without preparation, in any suitable animal, a good crop of artificial tuberculosis through inoculation, in the course of a few weeks, showing that in this respect the virus of phthisis differs in no wise from other diseases which are inoculable.

There is abundant testimony to show that the hospital treatment of consumptives is far superior in results to the home treatment. Such patients make the most remarkable recoveries when they are treated in institutions especially adapted for their care, notwithstanding the fact that under these circumstances they are constantly immersed in, and breathe the bacilli-laden air with their fellow-sufferers. Dr. Dettweiler, a physician of the highest eminence in phthisiology, claims to restore permanently sixty per cent. of the cases which come to his institution in Falkenstein. Then, again, it is quite frequent to find physicians both in America and in Europe, who are sufferers from consumption, and who are therefore compelled to spend most of their lives in the mountain resorts of their native countries, laboring hard among the many consumptives who crowd these places, and yet they experience not the least incon-

venience from their contact with these people, but on the other hand, often regain exceptionally good health. Here, if anywhere, the bacillus should find that suitable soil for its growth of which we hear so much, but instead of this it shrinks from its prescribed duty, and demonstrates its impotence as a practical factor in the causation of pulmonary phthisis.

Food Infection. – The same objections which hold against the bacillus as a cause of phthisis when inhaled, also apply to it as a cause operating through the digestive tract. Laboratory experiments without number almost, have been undertaken to show that animals become tuberculous when they are compelled to eat tuberculous sputum, and from these experiments the deduction is made by some that the human family is in imminent danger of annihilation from eating and drinking tuberculous meat and milk. These deductions are, however, in perfect keeping with the loose method of thinking which characterizes nearly everything that pertains to the question of the origin of phthisis at the present day. They are one-sided and extremely deceptive; for feeding experiments have rarely, and perhaps never, been successful in the case of dogs, and according to Johne,* more than half of the experiments failed which were made by him on rabbits, Guinea-pigs, sheep, goats, etc. More

* Johne, Dr. A., Die Geschichte der Tuberculose mit Besonderer Berücksichtigung der Tuberculose des Rindes, etc., Leipzig, 1883.

recently Kastner,† expressed the muscle juice of animals who died of tuberculosis and injected 1 ccm. of the same into the abdominal cavity of each of sixteen Guinea-pigs, under antiseptic precautions, without obtaining a single positive result. What justification is there then for the theory that human beings become tuberculous through their food, when it is shown that even the forced feeding with infected food succeeds in lighting up the disease only among certain species of the lower animals, while it utterly fails in others? Would similar logic be tolerated in any other department of medicine? Now what do the hard dry facts of experience say in regard to this question? Do they indicate that meat and milk are specially liable to give rise to phthisis? If this is true, then those who partake most of these foods should have the highest mortality from it; yet it appears that those who live on vegetable diet are as susceptible to it as those who do not. So also in the case of milk. If this produced any special liability to this disease then those who chiefly subsist on it should also display the greatest degree of susceptibility to it. Statistics demonstrate that only one-twelfth as many deaths occur from this disease, between the ages of from one to five—the natural milk-drinking period—as between

†W. Kastner, Experimentelle Beiträge zur Infectiosität des Fleisches Tuberculöser Rinder. Münchener Med. Wochenschrift, 1889, No. 34.

the ages of from twenty to forty—during which period comparatively few drink milk. In relation to this Dr. Hiller states (*Lancet*, July 4th, 1887, p. 1141), that out of a total *post-mortem* examinations of the bodies of thirteen hundred infants and children he only met with one case of tuberculosis. The same holds true when inquiry is made into the relative frequency of tuberculosis in old and young animals of the bovine species. The statistics of the Berlin abattoir show that among 320,000 calves three were only 17 tuberculous; but among 398,000 cattle there were 8,000 that were tuberculous.

Isolation.—History repeats itself in medicine as it does in society. The drama of isolating consumptives, and of guarding against their contaminating influences, which is receiving serious consideration at the present time in some of the principal cities of Europe and of this country, has been enacted before with all its attendant horrors, but the lesson which it should teach is recklessly disregarded. If phthisis is propagated through contagion, *i. e.*, if it is maintained through personal contact, then segregation is the only available remedy. One follows the other as naturally and as logically as night follows day. Over one hundred years ago the inhabitants of Naples reasoned in the same way, when in 1782,*

* Die Tuberculosenfrage vor Hundert Jahren. von Dr. J. Uffelmann, Berliner Klin. Wochenschrift, 1883, S. 369.

they introduced and enforced the most extraordinary and vigorous laws that have been enacted for the suppression of any disease.

At that time the medical advisors of the Board of Health of Naples, among whom were the most talented physicians of the day, became convinced that pulmonary consumption was a highly contagious disease, and with a view towards exterminating it they issued the following decrees which went into effect July 19th of that year:

(1.) Every physician was compelled to report every case of pulmonary tuberculosis, "l' ulcera pulmonale," under a penalty of a fine of 300 ducats ($180.00), or imprisonment for ten years.

(2.) Poor consumptives were sent at once to a hospital.

(3.) The hospital management was compelled to keep separate all clothing or linen used for or by such patients.

(4.) An inventory of all clothing belonging to patients was ordered to be taken immediately after his reception in the hospital, as well as after the patient's death. Any infringement of this law was punishable with imprisonment.

(5.) That portion of the patient's furniture which was not in immediate contact with him, was ordered to be thoroughly cleansed, and that suspected of infection was to be burned.

(6.) The rooms of patients who died in their own homes were ordered to be thoroughly disinfected, the ceilings, walls, and floors to be renewed, the doors and windows to be burned and new ones to be substituted.

(7.) Dwellings thus renewed were not to be inhabited for one year.

(8.) Heavy fines were to be imposed on all who bought and sold clothing belonging to phthisical patients.

These laws, which placed pulmonary consumption on a level with small-pox, produced a powerful impression wherever their practical application became necessary. As may well be imagined, the family with phthisis within its fold was regarded as one of the most unfortunate institutions on the face of the earth. The patient himself was looked upon as a public pest, and his relatives were shunned, and when poor, were driven to want. Houses in which patients died with the disease came into disrepute, and many of their owners were turned into beggars. In spite of such a desperate and pitiable condition of the people, and of the growing opposition of the medical profession, the government enforced all these laws until the year 1809, when, owing to some doubts on the part of the latter concerning the feasibility and wisdom of upholding such stringent and oppressive measures, the medical faculty was again consulted as to its belief concerning the contagiousness of tuberculosis. At this time, owing to a marked change of feeling in the minds of the medical men belonging to the Board of Health, the majority decided against the contagiousness of this disease, but the older members who had the greatest influence with the government, still held to its contagiousness, and, although in the minority, they had sufficient power to maintain these laws until about 1848, when, on account of their ineffectiveness, they became obsolete.

Testimony to the same effect comes from Dr.

Valentin (*Voyage Médical en Italie, etc.*—Nancy et Paris, 1822; Henke's Zeitschrift. Erganzungsheft, 5, 1825, S. 288), who says that in Naples and Rome phthisis is regarded as contagious. If a person dies in a private house, of this disease, not only are the clothing, bed and bedding, and the furniture, which were in use by the deceased, burned, but the plastering, floors, doors, and windows are all torn out of the room in which he died. Dr. Robert Sim (writing from Naples, *Medical Times and Gazette*, 1860, vol. i, p. 363) states that in this city the belief that phthisis pulmonalis is infectious—very highly infectious—is so prevalent that when a person dies of it, the furniture of the room occupied by the sick, together with the bedding and bed, is immediately burned. This belief is not confined to the ignorant Neapolitans, but is shared by the inhabitants of the palace; and it is not a harmless one, because it leads to the neglect of the sick, and even to worse consequences. "A member of the royal family some time ago was sent to die in a 'segregated house' in the country. I need not remark on the inhumanity of sending persons who are in the last stage of this disease to die away from their friends."

Now what has been accomplished by these laws, during and since their enforcement? Absolutely nothing but needless suffering. Professor de Renzi says (*Storia della medicinæ Italia*, p. 512) that the injury which has been inflicted on Naples is simply

indescribable. Did their influence diminish the death rate from phthisis? According to Valentin (loc. cit.), one-fifth of the deaths in Naples and in Rome were produced by phthisis in 1822. In Corradi's *Phthisis in Italy* (*British and Foreign Medico-Chirurgical Review*, vol. xlv, page 112) it is stated that so far as Naples is concerned, Drs. Achille Spatuzzi and Luigi, who have paid a great deal of attention to the mortuary returns of that city, affirm that a sixth or a seventh of the whole mortality was due to pulmonary phthisis in 1860, and another sixth or eighth to scrofula and rachitis; and Enrico di Renzi says, in a competition essay on the alimentation of the lower classes in Naples (1863), that this city is fully as liable to phthisis as either London or Paris—one-eighth of its deaths being due to this disease, and consequently its death-rate from this source surpasses that of Turin or Milan, where there is about one-twelfth.

These historical facts and figures will have to be met by those who claim that phthisis pulmonalis is a contagious disease. In this city of Naples, a plan of prevention was carried out with that thoroughness which can only be done by the machinery of a centralized Government, and one which would scarcely be tolerated by the social institutions of the present day. It is useless to plead that a knowledge of the bacillus gives us more power, and will enable us to prevent the disease more effectively than the peo-

ple of Naples did for sixty-six years without such knowledge. The authorities and the people of that city were as firm in their conviction that phthisis was a contagious disease, as the most enthusiastic bacillus theorist is, and they carried out their ideas of prevention with the utmost exactness and caution. Indeed, the requirements of the modern contagionists for stamping out phthisis seem more like the vaporings of a child's brain than the outcome of sober reflection, when compared with the strenuous efforts which these people put forth to crush out this disease. It was not sufficient for them, as it is for the former, that consumptives should cease to spit in handkerchiefs, on floors, ground, and pavements, and use spittoons instead; but these people were at once separated from the well, and in all probability neither they, their sputa, nor their bacilli, ever came in contact with the outside world again. The clothing and furniture in use before their segregation, was burned; the dwellings in which such people died were renewed, and were not allowed to be inhabited for one year. Barbarous and inhuman as these measures were, they must be regarded as nothing but the logic of the contagionists' premises carried to a consistent and relentless conclusion, and, as we have seen, with the most lamentable results. They not only inflicted an enormous amount of injury, but they were powerless to stay the death-rate from phthisis in the least; in fact, statistics show that this was, during and since the ex-

istence of these laws, at least as high and probably higher than in cities where segregation never received any consideration. Laws of a similar character were in operation for some time in Portugal, and from all accounts, were followed by the same consequences. In view of these developments, does it not seem a strange satire on history to find men who are again willing to repeat and to enforce the follies of a hundred years ago?

CHAPTER XV.

WHO ARE MOST LIABLE TO CONSUMPTION.

Influence of Heredity.—So far it has been found that it is not those who are most exposed to the influence of the tubercle bacillus who become most liable to phthisis. Up to the time of the discovery of this micro-organism there existed a general belief that heredity played a most important rôle in the production of pulmonary consumption, but since then it has been found that this conviction, which was apparently so firmly established, rested on an insecure foundation, and its value as a determining feature in this disease has, therefore, very seriously depreciated in the minds of the believers in the contagion theory.

At the very outset of this chapter it may be stated as a truism that he who denies heredity in pulmonary consumption might as well undertake to deny the existence of the disease itself. One is no more illogical or utopian than the other. Any one, only partially familiar with the laws of organic nature, must know that that which is acquired during the life of the individual is infinitessimal when compared with all the qualities which are inherited. That which holds true in life or in health also holds true in disease.

Now those who refuse to accept the hereditary

nature of phthisis, nominally fall into two classes: (1) Those who realize the incompatibility of contagiousness and heredity in the same disease, and who insist that all possess an equal liability; and: (2) Those who recognize that the children of consumptive parents are more prone to die of the same disease, but who by a little sophistical strategy convince themselves that the difference in susceptibility is acquired and not inherited. My friend, Dr. Lawrence F. Flick, of this city, is, I believe, one of the warmest exponents of the latter doctrine, and in order to avoid misunderstanding, I take much pleasure in copying a few paragraphs from his, in very many respects, interesting address,* on "How to Take Care of the Lungs." On page 5, he says:

Hereditary disease does not exist in the true meaning of the word hereditary. That children are born with diseases which they got from their parents cannot be denied, for it is a matter of frequent occurrence. To inherit a thing, however, presupposes a time when the thing was not possessed, and these children have the disease at the time of birth. I deny that a child can be born perfectly healthy in all its organs, glands, and parts, and years thereafter develop a disease which it got from its parents, or what is still more absurd, carry the disease dormant throughout its life, and transmit it to the next or the second succeeding generation. It

* Delivered before the Alumni Association of the Philadelphia College of Pharmacy, at its Fifth Annual Meeting, February 12th, 1889.

needs but very little knowledge of physiology and a moment's reflection for a thinking mind to relegate this theory to the realms of medical superstition. . . . . The real explanation of the phenomena so frequently observed, of whole families dying of consumption, is to be found in predisposition. Predisposition is a peculiar state of the body, either because of the development of its parts, the condition of the blood or the working of the nervous system, by reason of which it is prone to develop certain diseases.

If Dr. Flick's argument were not specious, all that he says concerning the absurdity of the hereditary theory of disease might be accepted. But what warrant has he for assuming that children are born perfectly healthy and that the seeds of disease cannot be transmitted on account of such perfect organization? Does he not know that life is the product of an adaptation of the internal to the external; that the degree of life varies with the degree of adaptation; that disease and death are evidences that the adaptation is not completed; and that the millennium when we will be born and live perfect, both in body and in mind, will not appear for some time to come? The fact that the transmission of such imperfection is not demonstrable to the senses does not in the least argue against the possibility of its existence. Darwin says:

They who attempt to belittle the force of inheritance have not attended to natural history. The breeders of animals would smile at such simplicity; and if they condescended to make any

answer, might ask what would be the chance of winning a prize if two inferior animals were paired together? Why have pedigrees been scrupulously kept and published of the Shorthorn cattle, and more recently of the Hereford breed? Is it an illusion that these recently improved animals safely transmit their excellent qualities even when crossed with other breeds? have the Shorthorns, without good reason, been purchased at immense prices and exported to almost every quarter of the globe? * * * Hard cash paid down, over and over again, is an excellent test of inherited superiority. In fact the whole art of breeding, from which such great results have been attained during the present century, depends on the inheritance of each small detail of structure." *

The same illustrious author also cites many examples of inherited disease. He says (p. 16, *ibid*):

With gout, fifty per cent. of the cases observed in hospitals are, according to Dr. Garrod, inherited. * * * Every one knows how often insanity runs in families; and in some cases where several members of the same family, during three or four successive generations, have committed suicide. Striking instances have been recorded of epilepsy, consumption and asthma being inherited.

He also refers to inherited diseases of the eye, like drooping eyelids, hypermetropia, myopia, cataract, absence of the iris, opacity of the cornea, day-blindness, Daltonism, and points out that the law of inher-

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* Animals and Plants under Domestication, vol. ii, pp. 11 and 12.

itance not only applies to diseases of the human body, but also to those of animals.

How, then, are all the facts of inheritance to be accounted for on the score of a predisposition which developes during the life-time of the individual? If whole families die of a certain disease, why is it that the conditions which generate such a predisposition operate principally on families whose ancestors were affected with a similar disease? Why not to an equal degree on other families not burdened by the hereditary taint? This is an anomaly which seeks an explanation at the hands of those who pin their faith to the preposterous theory of predisposition without inheritance.

It is self-evident, as has already been intimated, that a contagious disease under the control of the law of inheritance, except in so far as this may lend protection, is an exceptional occurrence. A contagious disease must be free to attack every individual who is not already protected. Experience teaches that the children of those who have had small-pox are not as susceptible to the same disease as the children of those are who never had it—showing conclusively that the true contagious disease not only protects the individual but also his progeny. Is this true of phthisis? This disease does not only fail to protect the individual from future attacks, but really increases his liability and that of his descendants in the same direction; and I now propose to shew that a large number of consumptive cases fall within these lines.

In estimating the potency of heredity in pulmonary consumption, we must take into account that statisticians differ in their results because they include various degrees of relationship. When parental influence alone is considered, the percentage is lower than if that of the whole family is embraced in the calculation. "The 1,010 cases of the Brompton Hospital Report included only parents, and gave an average of 24 4 per cent. Dr. Fuller's 385 cases embraced grandparents, uncles and aunts, and furnished 59 per cent. Dr. Cotton's 1,000 cases included parents, brothers and sisters, giving 36.7 per cent., and Dr. Pollock's 1,200, similarly estimated, showed 30 per cent., while our own 1,000 cases shewed a percentage of 48.4 on the same basis." (Williams, *Pulmonary Consumption*, p. 62) Homann estimates it at 70 per cent., Russ at 83 per cent., while Dr. Schnyder (op. cit.) investigated the whole family influence in 3.461 cases of consumption, and found it present in 66.03 per cent.

Moreover, in connection with this subject, I take pleasure in referring to the admirable report of *Mortuary Statistics of the Mutual Life Insurance Company of New York* for 1877. On page 72 of this work are tabulated the family records of 1,031 cases of consumptives, and an equal number of cases of non-sumptives. This comparison shows that among the former there were one hundred and ninety-four, and among the latter one hundred and two family-mem-

bers who were tainted with the disease. In commenting on these data, the author says: "These figures sustain the general opinion regarding the influence of hereditary and family predisposition, in causing consumption, more fully than any hitherto published, and we consider them as affording positive proof of its correctness. We find that of an equal number of consumptives and non-consumptives, similar in every respect, nearly twice as many of the former had consumptive blood-relatives as that of the latter—18.81 and 10.89 per cent. respectively. The difference is too great and constant through all the subdivisions, and drawn from too large a series of cases, to be the result of chance, and we must consider it the expression of a law."

These data demonstrate very conclusively that more than one-half of those who die from consumption belong to families in which the disease previously existed. But if any further proof is necessary to substantiate the correctness of this view, it can be found in the contribution of Professor Langerhans (*loc. cit.*, p. 135), who had the rare opportunity of investigating the death rate of children who were born of healthy colonial parents, as well as that of those who were born of consumptive parents, and who came to Madeira for the purpose of restoring their health. The investigation was confined to those who were born between 1836 and 1864, and it extended to the year 1883; and it will be observed that both classes were

living in the same surroundings, and that all their conditions were practically alike, except those which were inherited. During that period there were born of healthy parents one hundred and forty-seven children. One of these died from phthisis, but, as has already been stated, he was strongly addicted to alcoholic abuse, aud contracted the disease while absent in England. During the same period there were born of consumptive parents one hundred and six children, eight of whom died, and eight are suffering from the disease, making sixteen in all who became phthisical before 1883. The data on which these figures rest are not very large, but the deductions which naturally spring from them are all the more valuable, because they deal with very few, or no intangible quantities.

Having now established beyond a doubt that pulmonary consumption is inherited in a large proportion of cases, let us also inquire into the nature of this influence, and find out what it really means. Heredity does not necessarily mean a transmission of the disease direct, but merely the imparting of a certain tendency by the parent to the offspring, which produces in the latter a greater susceptibility to the disease than that which exists in those who are born of non-consumptive parents. A process of this kind obviously implies the existence of two factors: (1) An influence; (2) a medium upon which this innfluence operates and impresses itself more or less perman-

ently. Now it goes without saying that all organic tissues are capable of registering impressions, but of all these the nervous system is the one which is pre-eminently endowed with the property of receiving, transmitting, and recording impressions. This marked sensitiveness of the nervous system not only explains why so many of its diseases assume a chronic form, but also why such affections are more readily transmitted from generation to generation, than those which involve other textures. In all diseases of the brain and nervous system, like insanity, epilepsy, hysteria, locomotor ataxia, neuralgia, diabetes, etc, inheritance plays a most essential rôle; but genuine contagious diseases, like small-pox, scarlet fever, measles, typhoid fever, cholera, etc., are not inheritable, because they invade tissues whose power of recording and transmitting impressions is too limited. It may be noted in passing, that while the transmissibility of the contagious diseases from parent to child is small, their reproductivity is great, because they involve tissues—the skin—the epithelial cells of which are specially endowed with the power of reproduction. Hence it is that diseases like syphilis, which implicates the cutaneous tissues, are communicable from person to person with comparative ease.

The hereditary nature of pulmonary consumption is therefore another strong proof of its nervous origin. The truth is, that in this, as in many other respects, its clinical and pathological course is so parallel to

that of nervous diseases in general, that its identity as such an affection, can hardly be mistaken. I have already shown that it is interchangeable with hysteria, that it is intimately associated with general paralysis, locomotor ataxia, multiple sclerosis, peripheral neuritis, etc., and the last paragraph develops the fact, that the reason why it is so easily inherited, is because it is a nervous disease. If this line of inquiry were extended further it would also show that about the same proportion of cases of insanity and of consumption are inherited (50 per cent.), and that, like in the case of consumption, insanity descends oftener from the mother than from the father.

Influence of Occupation.—It is the universal experience of the medical profession that in by far the largest number of cases, pulmonary consumption begins in the apex of either lung. The reason why the apex is more susceptible to this disease than the middle and lower parts of the lung is undoubtedly due to the defective expansion in that region; and this explains why our clerks, telegraph operators, tailors, shoemakers, and all those who follow in-door occupations, who habitually become stoop-shouldered and flat-chested, furnish such a large contingency to the army of consumptives. Such individuals are in constant danger of falling victims to the disease, though they are free from hereditary taint. This is especially true if their general vitality is depressed to a point, by the above-named cause, from which it re-

acts with difficulty against incidental disturbances like colds, malaria, and other diseases. Many consumptives trace their whole trouble to a cold, and although "having taken a cold" is a term to which there is attached but little definite meaning, it enters more frequently into common medical parlance than any other. A cold, as I take it, is chiefly a shock to the nervous system, in the chilling stage of which the peripheral, visceral, and vaso-motor nerves are markedly affected, the blood pressure rises, the surface of the body is cold, although the thermometer registers an elevated temperature. So soon as the primary effects of the shock have passed off, there follows a relaxation or paralysis of the same nerves, the blood again comes to the surface, and sweating more or less profuse, or some other flux may occur, after which the body returns to its normal condition again, providing, its reaction is vigorous. But if any portion of the body is already weakened the effects of the shock will be felt more keenly there than in any other area, and the strong tendency will be towards the establishment of permanent disease in this locality. In the lungs, under these circumstances, the product of the nerve-shock manifests itself, as it does in all organs with mucous surfaces, in the form of a catarrh, which usually selects the apex, because, as we have seen, the nutrition-tone of this spot is already lowered by reason of its previous functional inactivity.

That catarrhal inflammation is a common se-

quence of nerve affection is well demonstrated in trigeminal neuralgia where all the mucous surfaces which are supplied by this nerve, like those of the eye, nose, mouth, and tongue, assume the catarrhal condition, and there are cases on record in which the neuralgia of this nerve was of sufficient intensity to produce inflammation and ulceration of the cornea—a condition similar to that which has been produced by Majendie in the rabbit, by dividing the fifth nerve near the Gasserian ganglion.

Influence of the Order of Birth.—There can be no doubt that there is a direct antagonism between nutrition and reproduction, for "reproduction is a form of nutrition and a growth of the organism to a size beyond that belonging to it as an individual, so that a part is thus elevated into a new whole" (Haeckel, *General Morphologie*, Vol. II, p. 16). Herbert Spencer, in writing of the antagonism existing between growth and sexual genesis, says:

> Whether a deduction is made from one parent or from two, whether it is made from any part of the body indifferently, or from a specialized part, or whether it is made directly or indirectly, it remains in any case a deduction, and in proportion as it is great or frequent or both, it must restrain the increase of the individual (*Principles of Biology*, Vol. II, p. 428).

From these principles it follows that reproduction is most perfect when growth is completed and before bodily decay sets in; that the higher the rate of nutrition during the reproductive period the higher the rate

of reproduction; and that if the bodily expenditure is greater for other purposes than for reproduction there is a corresponding deduction from the resources which would otherwise be devoted to the maintenance of the latter process. In other words, offspring will be, if other things are equal, most vigorous when the growth of both parents is complete and the state of nutrition is good; and least vigorous and most prone to decadence when the state of nutrition in the parents is low, when their expenditures for other purposes is great, or when reproduction takes place rapidly or follows at short intervals, or takes place before the body is fully developed, or during the period of bodily decline. All other things being equal then, the last or the youngest children, as well as those who are born before the bodies of the parents are fully developed, should be the weakest, and those born during the remainder or the middle part of the reproductive period should be the strongest.

In Dr. Brehmer's great work on *Die Aetiologie der Chronischen Lungenschwindsucht.* are recorded the histories of four hundred cases of pulmonary consumption which afford a striking confirmation of the correctness of the principles mentioned in the last paragraph. I have taken the liberty of analyzing these cases and obtained the following results:

1. The members of large families are less vigorous than those of small families.

2. The youngest members of numerous families, pro-

vided their parents are healthy and come from small families, are most liable to pulmonary consumption

3. Both the youngest and the oldest members of small families are liable to pulmonary consumption, while the intermediate members are less susceptible to it, even though the parents and grandparents were healthy and became aged; provided, that either or both parents come from the youngest members of numerous families.

4 Children born of parents having phthisical antecedents, are subject to the same law of liability as those children whose parents are healthy, but come from the youngest of numerous families, with the exception that it is not necessary for one or both parents to come from the youngest of their families.

5. Those children who are born within a year after the birth of the preceding members are more liable to pulmonary consumption than those who are born two or three years apart.

6. Children born of phthisical parents are liable to pulmonary consumption three years earlier on the average than those born of healthy parents.

7. Given a consumptive coming from a numerous family, the parents of which came from medium sized or small but healthy families, we can predict that the patient is the youngest or one of the youngest of the family; or given a consumptive coming from a small family, the parents of which came from numerous families, and were among the youngest but healthy, we can predict that the patient either belongs to the youngest or the oldest, and is not one of the intermediate ones of his family; or again given a consumptive coming from a small family, the parents of which came from a numerous family, and who have a phthisical history, we can predict that the patient belongs to either the older or the younger members, provided the patient's parents are not the

youngest of their families; if they are the youngest, then the intermediate members are not exempt, although even then more so than those of either extreme.

If the inquiry as to who are most liable to pulmonary consumption be pushed still further it will be found that the male is more prone to it than the female; that those living near the sea level more than those living on elevations; that immigrants are more than residents; that the full blooded Indian more than mixed, etc.; but I think enough has already been said in this chapter to show that it is not a capricious disease, and that it selects and attacks its victims with a regularity that is as inflexible as it is relentless.

CHAPTER XVII.

SUMMARY AND CONCLUSIONS.

In summing up the arguments which have been developed in these pages, it is obvious that the controversy whether phthisis is a contagious disease or not rests on the broad principle whether it originates from within or from without the body. Among the self-evident corollaries which flow from this principle, are: (1) That any disease, no matter what its nature may be, if it originates from the outside, must spread among those who are exposed to the cause or causes which give rise to it; and (2) that segregation has the power of checking and of eradicating diseases which arise from causes outside of the body.

Now if pulmonary consumption is a germ disease and arises from without the body, how does it stand in regard to the first corollary? Tubercle bacilli abound in hospitals for consumptives, and in the homes of those who suffer from the disease, yet no one can show that the visiting and resident physicians, nurses, and attendants of such hospitals are more liable to consumption than those living outside; nor are physicians in private practice, who constantly come in contact with the disease, as liable to the disease as are butchers, stone-grinders, etc., who are subject to very little or no exposure of this kind; nor can it be shown that there is any contagion between

husband and wife when either is affected; nor is there any testimony whatever to show that infection occurs through houses or food.

When we inquire how pulmonary consumption, as a germ disease, answers to the second requirement, it will be seen that in localities where segregation of the strictest kind was practiced for a long time, not the least diminution in the death-rate from this disease occurred, but, on the other hand, a great deal of direct and indirect harm had been inflicted by the enforcement of the measure.

Apparently there is a conflict between these practical results, and the findings of the pathological laboratory. For the believer in the bacillus as a cause of the disease will say that this organism has been isolated by cultivation, and produced tuberculosis in animals which were inoculated with it, and this is sufficient proof for him to believe that pulmonary consumption is always propagated from man to man by inoculation of this germ. The formidable nature of this argument will dissolve on closer inspection; for, as has already been stated, the natural genesis of a disease is a very different thing from its propagation by inoculation. There are very few, if any, diseases which are not associated with specific germs, and which may not be communicated by inoculation, but the product of such infection throws no light whatever on the natural evolution of any disease. Inoculation is an artificial method of communicating dis-

ease, and it finds its perfect analogue in the practice of grafting in the vegetable world. It is well known that a graft is capable of communicating the peculiar properties of the fruit and of the variegated leaf-colors of the tree or plant from which it is taken, to the whole tree or plant on which it is grafted; but this phenomenon leaves us entirely in the dark regarding the place, conditions, and surroundings, in and under which the fruit and the leaf-colors of the tree or plant which produced the graft were brought forth. These properties are evidently the result of a long line of growth and development, but the contagionist might with equally good grace argue that because they are capable of being transmitted in this artificial manner, they are under all circumstances the products of inoculation. The absurdity of this mode of reasoning is self-evident, however, to every one.

Laboratory experiments, if rightly conducted, therefore, confirm the researches of clinical medicine, inasmuch as they show that phthisis which originates through a long line and a multiplicity of causes independent of contagion, may be reproduced artificially in a comparatively short time by a method of grafting known as inoculation. A disregard of this relation has led to many hygienic and therapeutic blunders of the worst type, and has been the cause of much misdirected energy, which, under the guidance of a more rational ætiology, would have advanced phthisio-therapy to that higher general plane which it should occupy to-day.

Let us now endeavor to see how the theory of consumption as a neurosis harmonizes with the facts and statistics which are known and which have been collected in this essay concerning this disease. This theory implies that consumption is a product of vagus disease, and hence, if the theory is tenable, it must be shown: (1) That vagus disease produces consumption, and: (2) That a distinct connection exists between consumption and the influences which are known to produce disintegration of the vagi. To demonstrate this, several possible avenues offer themselves. First, by experimentation on animals; second, by finding out whether in human phthisis there is any evidence of vagus disease, and whether the latter sustains a causal relation to the former; and third, by ascertaining whether the influence which produces degeneration of the vagi and of other nerves also produces phthisis in the human subject.

First: While experiments on animals teach that pneumonia and even phthisis may be produced by section, and irritation of the vagi without division, it is very evident from what appears in these pages that phthisis is a disease which is not dependent on a transitory, but on a protracted and long-continued disorder of the vagi, and that hence it is very difficult to regulate the necessary degree of irritation in the vagi of animals to secure the desired results. On account of this difficulty coupled with the obvious objection of infection which may be brought against such ex-

periments, I abandoned this plan for the more serviceable and practical one of demonstrating the truth of the neurotic theory on the human organism itself.

Second: In scanning the literature of phthisis and nervous diseases, I found *eighty-one* cases of phthisis in which vagus disease existed. Now, is this a mere coincidence or is there evidence that the vagus disease bears a causal relation to the pulmonary affection? A decision in regard to this is reached by analyzing these cases. Some of them (29) show as clearly as anything can be shown that the phthisical degeneration was the result of a primary local disorganization of the vagi due to the presence of aneurisms, tumors, enlarged glands, etc., on these nerves. Here we possess all the necessary requirements and conditions of a perfect experiment—such as we aim to get in animals; and unless we assume that the phthisis preceded the local pressure on the vagi, and that the latter was induced by the former, both of which are highly improbable, and neither is confirmed by anything that appears in the histories of the cases, we are bound to accept that phthisis is a direct resultant of vagus disease.

In a number of other cases (20) vagus disease and phthisis were associated with various nervous affections such as multiple neuritis, tabes dorsalis, bulbar paralysis, neuromata, etc. In these instances there is again every reason to believe that the pulmonary manifestations were secondary to the nervous

disintegration. Again there are fourteen cases in which phthisis was associated with epilepsy and diabetes, as well as with disease of either the vagi or the medulla oblongata. In many and probably most instances epilepsy and diabetes arise in the region of the medulla oblongata, and hence, viewing phthisis from a neurotic standpoint, it is very easy to see why these three diseases should be so closely affiliated. Then, again, a portion of these cases (18) show that phthisis is the product of vagus degeneration brought about through the pernicious influence of alcohol and syphilis. These cases illustrate, then, beyond the cavil of a doubt that pulmonary phthisis is the product of vagus degeneration, but what must still be shown to make the chain of evidence complete, is that this view of the origin of phthisis corresponds with the facts and statistics which have been acquired through other clinical experience.

What then are some of the prominent features in the history of phthisis? One of these is its absence in barbarous, and its prevalence in civilized communities. This is so well marked that it has led very many good meaning professional men to believe that the disease was brought to the new country by its first settlers, and that it then spread through contagion. The erroneousness of this view, however, becomes very clear on deeper inspection, for both barbarism and civilization represent parts of the process of adaptation which is constantly going on between

the body and its environment. This implies that the human body by means of its various organs is constantly employed in a struggle with its exterior, that different organs are engaged in different phases of social existence; and that the organs which participate most actively in this struggle are in the greatest danger of abuse, overwork, and disease. To exemplify this it may be said that the savage is cold and unemotional, cares little for the morrow, supplies his wants, which are few, by hunting and fishing, and therefore his adaptation is chiefly maintained by the exercise of those functions which are common to animals and men, such as digestion, locomotion, procreation, etc., while the higher powers of the brain and nervous system remain more or less dormant.

But, on the other hand, civilization with its education, knowledge and inventions, its diverse manners and customs, its changeable institutions, its rankling politics, its innumerable arts, sciences, and manufacturers, its multiplicity of employments, its accompanying proneness to vices, excesses and abuses of all sorts, shows that its plane of adaptation is higher than that of barbarism, and that its maintenance on such a scale demands the exercise of the highest faculties in the possession of man. These reside in the brain and nervous system. In fact nothing but a high state of development of these organs makes civilization possible, and divides the latter from barbarism. The active participation of these

organs in the process of civilization renders them more liable to injury and disease, hence it is perfectly obvious that diseases of this tract of tissue must be much more common in civilized than in barbarous life. This conclusion is in entire accord with our knowledge of the prevalence of insanity and nervous diseases in this respect, for this shows that affections of this class attain their maximum among civilized races, and their minimum among savage races. It is thus found that the closest analogy obtains between insanity, and nervous diseases in general, and phthisis in particular, and if these facts are considered in connection with the other facts which have been brought forward in this essay it is equally clear that the reason why the latter disease should pursue a similar course is because essentially it is a nervous disease.

In regard to the direct causes of phthisis it must be concluded from what has been said that alcohol is one of the most potent. It has been plainly shown that it produces disintegration of the nervous system in general, and of the pneumogastric nerves in particular, and that through the latter influence it generates phthisis. More than this it has been shown that the effects of alcohol are transmitted to the offspring in the form of phthisis. Alcohol is, therefore, not only a powerful factor in the ætiology of this disease, but on investigation it will be found to play an equally prominent part in the production of insanity and nervous disease.

Syphilis is another direct cause of phthisis. Without going to that extreme which some authors are inclined to do who claim that phthisis and scrofula are merely attenuated forms of syphilis, I am prepared to accept that it is oftener present in phthisis than is commonly suspected. This is epecially true among the lower classes. It is also a recognized cause of insanity and nervous diseases. Among other direct causes of phthisis must be reckoned lead, mercury, arsenic, brass, the virus of measles, scarlatina, whooping-cough, diphtheria, etc., examples of which are given in the preceding tabulated records, and in fact any other influence which has a special disintegrating effect on the nervous system.

Now, although it is true that phthisis is almost entirely absent in primitive social existence, it must not be forgotten that when savagism first comes in contact with dominant civilization, its death rate from phthisis increases and often rises to startling proportions. This augmented liability to phthisis is partly due to the fact that the savage is suddenly thrown into and overawed by an environment with which he has very little in common, and has very little power to bring himself in harmony with it. He is like an animal on which is imposed a new condition incidental to a succession of geological changes. If the change is not too abrupt, and it possesses the power of adapting itself to its modified surroundings, it will survive, if not, it will die. Hence, from what has already

been said concerning the nervous system as an instrument through which adaptation is effected in the animal world, it is perfectly clear that, in the case of the savage, under these circumstances the brunt of the battle devolves on this part of the body, and that through this his liability to phthisis is increased.

The augmented mortality from phthisis among savages, in such a transition stage, is also due to the fact that they fall an easy prey to the vices of civilization. It is universally admitted that alcohol and syphilis are the greatest curses which the savage receives from his white civilizer, and when we reflect on the potency of these agencies in generating phthisis, and on their importance as a factor in maintaining the ranks of the hundreds of thousands of civilized people who are annually cut off by this malady, we cannot help but believe that they are largely responsible for sowing the seeds of the same disease among these poor helpless people.

On recapitulating the evidence which has been offered in these pages, the following conclusions may, I think, be legitimately drawn:

1. That in all probability every disease possesses its attendant micro-organism.

2. That the natural genesis of a disease is altogether different from its artificial transplantation.

3. That the inoculability of a disease is not the least evidence of its practical contagiousness.

4. That the theory of the contagiousness of pulmonary consumption rests almost entirely on suspicions, and on

laboratory experiments which are unsupported by clinical facts.

5. That tubercle is not in itself a menace to life.

6. That there is no correspondence between the number of those who are exposed to the bacilli and those who contract pulmonary consumption.

7. That all therapeutic and hygienic measures which have been based solely on the bacillary origin of consumption are disastrous failures.

8. That consumption is inherited in about fifty per cent. of all the cases.

9. That a hereditary disease is not contagious unless it affects the nervous system.

10. That catarrhal phthisis may be produced in animals by section of the vagi.

11. That pulmonary consumption is a disease in which the vagi are primarily involved.

12. That alcohol and syphilis produce pulmonary consumption by inducing vagus disease.

13. That in all probability, arsenic, lead, mercury, brass, and other substances produce consumption by reason of their specific action on the nervous system.

14. That diabetes, beri-beri, leprosy, and probably lupus and pellagra, are intimately associated with pulmonary consumption, because fundamentally the evidence appears to show that they are nervous diseases.

15. That the neurotic theory of pulmonary consumption shows such a rational connection between cause and effect as no other theory does, and explains why nervous or mental shock, whooping-cough, alcoholism, syphilis, arsenic, lead, mercury, etc., produce this disease; why the latter is associated with insanity, with disease of the brain, spinal cord, and peripheral nerves, with diabetes, beri-beri, leprosy, pellagra, etc.; why it is inherited; why the youngest and the

oldest of a family are most prone to it; why segregation or quarantining is useless; and why the hypophosphites, cod-liver oil, electricity, and the maintenance of the nutrition-tone, are such invaluable aids in the treatment of this disease.

CHAPTER XVII.

THE THERAPEUTICS OF CONSUMPTION BASED ON THE NERVOUS ORIGIN OF THE LATTER.

The cardinal principle on which this work rests, then, is that pulmonary consumption is not a local but a constitutional disease; that it consists in an exhausted state of the nervous system in general and of the pulmonary nerves in particular. Exhaustion, therefore, being the fundamental morbid condition with which we have to deal, therapeutically, the question arises as to the best manner of combating this evil. A recent editorial of the Medical and Surgical Reporter (Sept. 7th, 1889), *On Rest in the Treatment of Pulmonary Consumption*, defined the consumptive's condition as one that verges "towards a state of physiological bankruptcy. The disease makes a fatal drain on his constitutional resources. With him it is a real living warfare between the forces of his body and those of the disease. The line of antagonism which divides these forces is neither hard nor fast, but constantly shifts its position in accordance with the ebb and flow of his vitality. When he is weak the disease advances, and when he is strong it recedes. The remedial indications are, therefore, towards a fortification of the constitutional resistance. This can only be accomplished by economizing the bodily

forces—that is, by diminishing the outgo and by increasing the income, and physiological rest is one of the most valuable adjuvants in securing this end."

In consonance with the view expressed here, I believe that rest, absolute or an approach to it, is one of the most vital points in the treatment of pulmonary consumption. The idea of rest, however, is contrary to the orthodox methods of managing this disease. Custom has handed down from time almost immemorial the dictum that exercise is the one indispensable consideration in its treatment, and I apprehend that, on account of this universal impression, the recommendations which I am about to make will be received with a feeling of cold indifference, or, perchance, with opposition. Notwithstanding the risks to which I thus expose myself, I am constrained to say, in the interest of truth, so far as I am able to see it, that I believe the prevailing opinion that consumptives must have plenty of exercise, is one of the greatest stumbling-blocks in the successful management of this disease. So deeply-rooted is this idea in the mind of the public, that patients persist in walking out until their vital energies are totally exhausted, when they are compelled to give up and go to bed, and in most cases they go there to die. This is no fanciful drawing, and it pathetically illustrates the truthfulness of the familiar expression, "that a consumptive never goes to bed for good unless it is to die."

Patients often urge the necessity of exercise in order to cultivate an appetite. They say if they are kept quiet they are unable to eat. This illogical deduction is made from what they know exercise is capable of doing in a healthy state, when, it is true, muscular activity produces a luxurious appetite. But it is also true that even in the best of health the body can become so thoroughly fatigued through physical exercise that the appetite disappears entirely, and returns only after a short period of rest, during which the function of the stomach becomes reinvigorated. Such a condition of fatigue is an approach to that which obtains nearly constantly among consumptives. If their bodies are not in a state of chronic exhaustion already, they become so on the slightest exertion, and the appetite disappears because the stomach, in sympathy with the whole body, is too tired and too weak to carry on the process of digestion. It is perfectly plain, then, that by exercising, such patients defeat the very end they have in view. My experience has made me familiar with cases of this kind who were troubled with absolute anorexia so long as they persisted in taking even moderate exercise, and whose appetite reappeared, and rather vigorously too, so soon as they were placed at rest in bed.

I do not by any means wish to be understood that I disapprove entirely of exercise in the treatment of this disease. Exercise is undoubtedly beneficial to those who have gained considerable strength from

treatment, but I contend that it weakens those who are weak already, as much as it strengthens the strong. Probably this whole question of rest and exercise in consumption can best be practically and briefly illustrated by taking an example from the field of finance. It is an old and a true saying, that money makes money. A man who has a certain amount of capital can make money much more easily than he who has none or very little. If the latter spends as much as he takes in, his finances will be in a crippled condition all his life; but if he halts that is, if he diminishes his outflow, and maintains or increases his income—his capital will accumulate, and in time he will be able to compete with other capitalists. So the taking of exercise which is to be beneficial implies pre-existing strength, and by putting this strength out at proper interest, or to proper use, it will grow and accumulate; but the consumptive who has little or no extra strength at the outset, must reduce his expenditure or enlarge his income, or else he will be compelled to go into physiological insolvency.

In pursuance of this principle, I have of late prohibited my patients from walking, and if necessary I insist on their going to bed, or on remaining in the recumbent posture, during at least seven-eighths of the twenty-four hours. Those who become tired on the slightest physical exertion, must remain in the horizontal position constantly for a month or six

weeks, or until their condition warrants a change in posture. This plan of treatment does not imply confinement to the room, or even to the house, but, if able, the patients are allowed to walk out and sit quietly in the open air, or, if too weak to walk, they are carried out, and permitted to remain there during the greater part of the day—being, of course, warmly and suitably clad, and well protected from any unfriendly draughts of wind. They will often rebel against this protracted rest merely because they do not realize the proper nature of their condition; but if this is once fully comprehended, there are very few who will not appreciate the value of the proposal, and become willing to carry it into practical effect. They can be confronted with the argument that in such a persistent disease the question of getting well resolves itself into one of an accumulation of strength; that this point is gained much more readily if the whole body is kept quiet and in a recumbent or in a semi-recumbent position, when both the nervous and muscular systems are as near perfect rest as they can get during the waking hours.

Dr. A. Volland, of Davos, in his work on *Die Behandlung der Lungenschwindsucht im Hochgebirge*, says (p. 18) that rest in the open air is the first duty of the patient. If able, he is allowed to sit out of doors, and if not he is confined to bed in a well ventilated chamber. Both Dr. Brehmer and Dr. Detweiler, in their published works on the treatment of consump-

tion, advocate and enforce rest as an essential factor in the treatment of consumption in their private institutions.

The quietness of the patient having been secured, the next thing of importance to occupy the attention of the physician is the elevated temperature. The fever of pulmonary consumption, like the pyrexia of other diseases, is the result of a disturbance in the heat-regulating centre of the nervous system—and it must therefore be regarded as a nervous phenomenon. The degree of fever is always an excellent guide in determining the degree of nervous disturbance which exists in any given case of pulmonary consumption. The intensity of the former is not always indicated by the physical signs. Indeed, the very gravest forms of the disease come before you with very little evidence of pulmonary disintegration, in which the thermometer shows an evening temperature of perhaps 104° Fahr., with chills and a subnormal temperature in the morning. Such cases are living illustrations of the truthfulness of the neurotic theory of pulmonary consumption. The chills, the high fever, the sweats, the aching pains in the limbs and in the back, all indicate a grave disturbance of the nervous system, while the respiratory organs are comparatively free from any signs of disease. Again, the degree of fever is not only a significant indication in regard to the severity of the disease, but it is an important aid in foreshadowing its course. If the

high fever persists in spite of the treatment, it serves to show the rebellious nature and the probable hopelessness of the case. It may be laid down as a certainty that no consumptive can get well, and maintain a temperature of 101° or 102° Fahr. at the same time. Of course it is altogether impossible to expect the fever of this disease to subside at once, but that which is meant to be impressed here is that the physician ought to have the consciousness that he has the power of controlling the temperature, and of maintaining it in the vicinity of or below 100° Fahr. An evening temperature of this degree may remain for three or six months, or even longer, in perfect consistency with permanent recovery under most circumstances.

Nowhere does the value of rest in the treatment of consumption stand out more prominently, and find stronger corroboration than in its application to the fever process. The truth is that no physician does full justice to his patient who prescribes exercise at the same time that he is making efforts to reduce the fever. It is astonishing how readily the temperature of a consumptive flies up on the slightest exertion. I have seen it rise more than one degree of Farenheit's scale in consequence of a patient swinging dumb-bells for five minutes, or taking a short brisk walk. The facility with which the temperature of a consumptive is raised by physical exercise explains why many patients of this character improve so

long as they are kept quiet in their beds, or in their rooms, but disappoint both their physician and themselves when in accordance with the prevailing fashion they are given their freedom, and are even urged to take plenty of physical exercise in the open air. Of course this tendency towards a rise in temperature when the patient first begins to move about exists in every case of this kind; but this should be carefully watched, and if it rises to 100° Farenheit he should be remanded to his couch, or be restricted in his movements until he has accumulated sufficient constitutional stamina to bear the exertion.

Rest is a febrifuge more powerful than quinine or antipyrin, and its application is one of the secrets of successsul treatment of fever in consumption which obtains in the closed institutions of Drs. Brehmer and Dettweiler in Germany. That this good result is not chiefly due to the elevated location of their institutions, on which they both lay so much stress, is well shown by the fact that Dr. Volland (*op. cit.*, p. 48) in Davos-Dörfli, at an elevation of about 5000 feet above the sea level, and probably 4000 feet higher than the location of either of the above named institutions, is compelled to follow a precisely similar course in reducing the fever of his phthisical patients.

From this it does not follow, however, that rest is the only remedy for the fever of consumption. We had recently added to our materia medica a number of agents which possess a powerful controlling influ-

ence on this processs. As has already been stated, it is clearly demonstrated, and principally through the experimental researches of Dr. Isaac Ott, that fever results from a disturbance of the heat centres of the nervous system, and it has also been shown that antipyrin, antifebrin, and phenacetin, the most useful of these agents, suppress fever in virtue of their action on the nervous system. Although these three substances are closely allied in chemical composition, quite an extended experience with them convinces me that there is quite a marked difference in their utility. Antipyrin is followed by excellent results. I have seen the high temperature, cough, expectoration, and anorexia of chronic phthisis subside promptly under the use of antipyrin given in fifteen-grain doses twice daily, or seven and a half grains every four hours. But if administered continuously for two or three weeks it is almost sure to call forth a cutaneous eruption of the most itching character. If it is given alternately with either antifebrin or phenacetin this is avoided. Antifebrin has never produced such striking beneficial results in my hands as those which were obtained from antipyrin, besides being very liable to bring on blueness of the lips, nose, ears, finger-nails, etc., if given too long. Of the two, phenacetin is probably the most valuable. In doses of four or five grains, every three or four hours, it will keep the temperature near the normal line in most cases. If these doses do not bring about the desired results,

they may be increased to ten and even fifteen grains, although it is seldom that more than eight or ten grains are necessary. This drug can be given for a much longer period than the other two, before it begins to manifest its toxic properties—in fact, I have never seen any bad results following its administration in the above-named doses. The antipyretic action of these agents tells only of a portion of their usefulness. They also exert a powerful tonic influence on the nervous system, and do as much good in this as in the former capacity. For this reason they should be continued after the fever has subsided, although in diminished doses.

Whatever else is done in the interest of a consumptive it still remains for food to perform the principle rôle in restoring his health. Food is the medium through which his physiological capital—the material to build up the tissues, and the force with which these are kept in running order—is increased; hence nothing taxes the ingenuity of the practitioner more, and nothing demonstrates his ability to better advantage than his success in dev sing ways and means whereby his patient is led to partake of a sufficient amount of nourishment. The want of eating, however, is peculiar to this disease. In many other wasting diseases the appetite, as a rule, remains the same, or improves, but in consumption it appears that the greater the emaciation the more marked becomes the anorexia. The stomach is, therefore, one of the chief

battle-grounds on which the destiny of the patient must be fought out, and it may be laid down as a law that, if a patient improves in every respect save in eating, he is still to be regarded as a non-convalescent.

Although the want of appetite shows itself so very early in the progress of consumption it is quite clear that this does not depend on a poor digestive power, but from what has been said already, it is probable that the ultimate cause of this derangement resides entirely outside of the alimentary canal. This view is also confirmed by the investigations of Schetty (Deutsches Archiv. für Klin. Med., 1889, S. 240) who, after testing the digestive function of twenty-five consumptives, in various stages of the disease, by examining the contents of their stomachs, in an hour or two after having received a certain quantity of food, concludes: (1) That the hydrochloric acid production is not diminished even in advanced cases: (2) That the power of albumin-digestion does nor vary from that of healthy persons, and: (3) That there is no impairment of the motor power of the stomach.

The dyspepsia of phthisis, is, therefore, not a local disorder and when the facts which support this view are connected with what has been said in regard to the significance of loss of appetite, and of waste and repair as symptoms of phthisis (p. 77), I think it will become manifest that this form of dyspepsia is dependent on general nervous apathy, and that it is

best treated by medicaments which act on the stomach indirectly through the nervous system. This theoretical consideration is confirmed by the practice of giving strychnine, nux vomica, atropine, quinine, antipyrin, and other agents which possess such a decided affinity for the nervous system, and which are considered so useful in restoring the waning appetite of this class of patients.

These developments indicate too, why it is that phthisical patients profit by being compelled to eat in accordance with the well known plan of Debove and others. On account of its difficult application, however, this plan is not practicable in most cases, and a great deal more can often be accomplished by a systematic method of feeding. A little food at regular intervals, and oft-repeated, will furnish a patient a large amount of nourishment in twenty-four hours, and will convince him that he has the power of ingesting a great deal more than he thought he had. I have seen patients who, absolutely refusing all kinds of food, by persuasion would take a glassful of milk, either cold or hot, every hour and a half or two hours, which gave them nearly two quarts of milk in a day, and would begin to partake of other foods so soon as their confidence in their digestive capacity was restored in this way.

An important feature in the dietary of a consumptive is variety. In this particular, of course more depends on the versatility of the cook than on

the physician, or nurse. She must be dextrous and able to render the bill of fare as tempting and as enticing as possible. The consumptive should not know in advance what the table has in store for him, so that he is not allowed sufficient time to manufacture excuses for not eating certain kinds of food. He always eats best when surprised by some new and inviting dish. He should also be permitted to eat anything for which his appetite craves, unless it is known to be positively injurious.

Now, what kinds of foods ought such patients to have? Clearly those which contain the greatest amount of food energy in the smallest bulk, and which at the same time confer the greatest amount of good on the body with the least expenditure of vital force in their digestion. Foremost among these are freshly expressed beef juice, beef, broiled, roasted, rare or scraped; roasted lamb or mutton; eggs, raw or soft-boiled; butter, cream, milk, oatmeal, beef-tea, soups, milk-punch, whisky, brandy, wine, beef peptonoids, beef extracts, malted milk, and other malt preparations. On the other hand, many other less nutritious foods and condiments, such as asparagus, lettuce, celery, green peas, tomatoes, potatoes, coffee, tea, chocolate, oils, spices, pepper, salt, vinegar, etc., should be admitted to the list. Meals should be served at regular intervals, five or six times a day, and the food should be slowly and thoroughly chewed before it is swallowed. Dinner taken in the

middle part of the day should be the heaviest meal, and the last at night should be the lightest one.

In feeding these patients, it is frequently found that the first meal in the morning is vomited. This is principally due to the violent coughing efforts which are required to expel the accumulation of catarrhal material in the lungs, and perchance in cavities, during the previous night. This annoying factor can be overcome in most cases by asking the patient to eat nothing until the paroxysm is over, or if it is necessary to eat, to partake only of milk, soup, brandy, or liquid food, and these in tablespoonful doses at short intervals.

Feeding by the rectum is a method which deserves greater consideration in the treatment of phthisis than it has so far received. In spite of theoretical objections, there is no question that, practically, the food which is placed in the lower bowel is digested and absorbed. This method not only economizes the strength of the stomach, but I have seen it, besides nourishing the patient, also counteract the tendency to diarrhœa which exists in many of these cases. For this purpose, milk, eggs, beef essence, Valentine's meat juice, the pulp of raw fresh beef, may be used. To the eggs and beef a small amount of pepsin may be added. The fluid foods are injected, while those of a semi-solid nature are best given in suppositories. One of the best ways to supply semi-solid food through the rectum is by hollow

suppositories large enough to contain half or a whole teaspoonful of raw-scraped beef pulp, and administered three or four times a day.

In addition to the measures which have been advocated in these pages, much good can be accomplished by giving cod-liver oil when it agrees, either by the mouth or rectum; by the administration of the hypophosphites; by massage of the whole body; by Galvanization of the pneumogastric nerves; by the application of iodine tincture to the neck over the course of these nerves; by the inhalation of oxygen and nitrous oxide and compressed air; by the practice of pulmonary gymnastics; and, in fact, by the employment of any agency which has the power of enhancing the local and general nutrition-tone of the body.